R.A.S.C.A.L.S.
RATS ARE SWEET CUTIES AND LOVABLE SOULS
We Love our Rats
The South African Rat Fan Club

Basic inheritance

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1 Introduction

In the rat fancy there are many varieties available, but how do we get all of them? And how does one breed a certain variety? Well, to answer the first question, almost all of our rat varieties are caused by mutations, which are the result of changes in the factors (of which there are many) that make up the normal or wildtype form of the fancy rat. The wildtype form is therefore a non-mutated variety and the wildtype of our fancy rats (*Rattus norvegicus*) is of course the standard ear agouti self (see figure 1). Dumbo ears are for instance a mutated version of the factor for ear shape in the rat (wildtype in this case is standard ears). To understand how all these factors work and how to breed certain varieties will therefore require a basic knowledge of genetics.

Figure 1: A standard ear agouti self female rat. When speaking in terms of genetics, we will refer to this variety as the wildtype form of our fancy rats. This variety is referred to as wildtype, not because it is a real wild rat, but because this variety is not mutated.

When mentioning the word genetics, many people think genetics are extremely complicated and cannot be mastered easily. Yes, going deeper into genetics will require knowledge of chemistry for instance, but for us as rat fanciers, we only need to know the very basics. In this article, I will try to explain genetics as simple as possible. However, this does not mean that this article can be read through in one go like a storybook. You will need to study each section in detail and make sure you understand it before moving on to the next section.

2 The cell and its reproduction

All cells come from other cells (Keeton 1973) and a new baby rat comes from the combination of the two reproductive cells obtained from his mother (her egg cell) and his father (his sperm cell). He also obtains his genetic characteristics from both his parents, so it should be clear that these genetic characteristics are somehow obtained from his parents reproductive cells (NZC 1994). This is why we are first going to look at the cell and its reproduction before tackling laws of inheritance.

2.1 Cell structure

A cell (see figure 2) is a tiny microscopic unit of which all living organisms such as ourselves, rats, plants, etc. are built up of. Rats are made up of billions of cells, but start of life as only one tiny cell known as the zygote (which is formed by the union of his mother's egg cell and his father's sperm cell). However, for this zygote to form and the resulting baby rat to grow into an adult, certain processes need to take place. This zygote need to divide and thus replicate itself to make two cells now, which also divides again to make four cells, etc., etc. This process is known as cellular reproduction of which there are two types, namely mitosis and meiosis, both of which we will look at more closely below (NZC 1994).

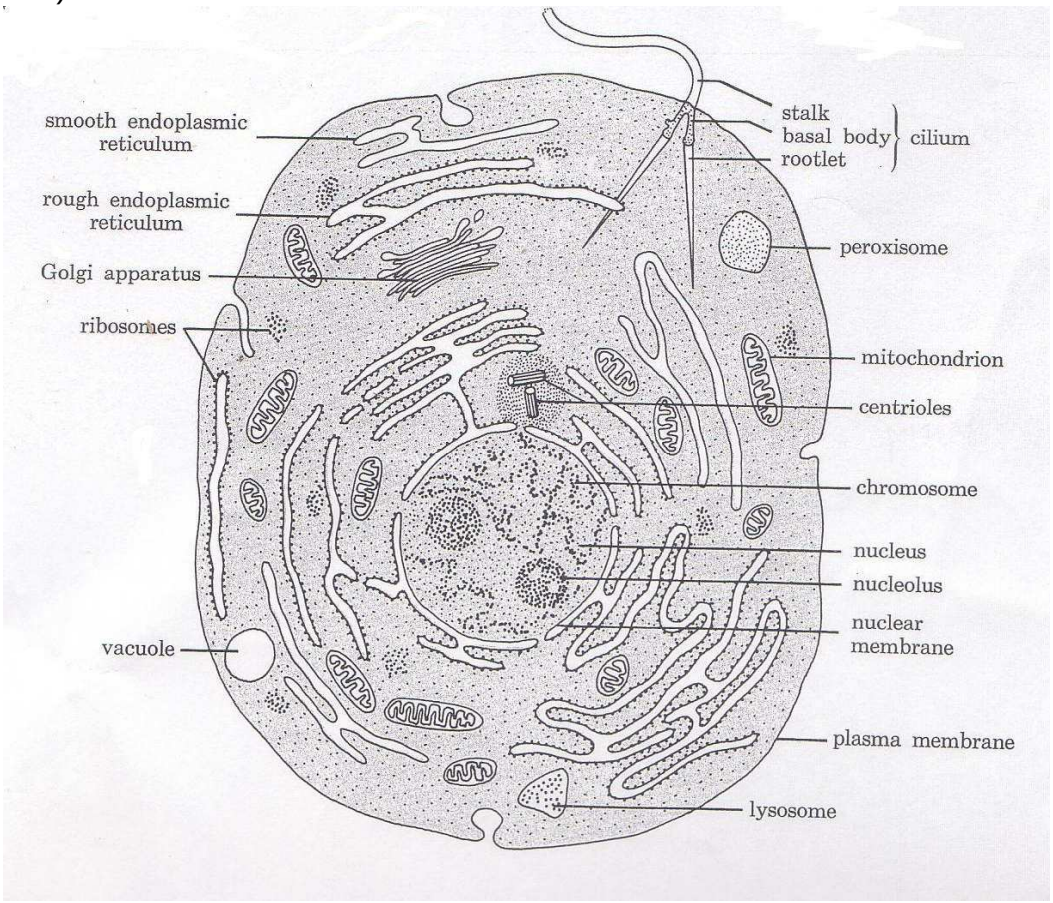


Figure 2: The structure of an animal cell (taken from Keeton 1973)

2.2 The nucleus and its chromosomes

In figure 2 we can see that the cell is composed of many parts, but the only part we are interested in here is the nucleus as it contains the genetic material or DNA (deoxyribonucleic acid). Each DNA molecule consists of two very long strands of four basic types of amino acids bonded together in sequence. Each strand of DNA fits into its corresponding strand almost like a zip and when the two strands are bonded together they twist to form a double helix structure. Because a DNA molecule is an extremely long molecule, it is neatly packed in a super-coiled structure known as the chromosome (Keeton 1973). Rats have 42 chromosomes (Wikipedia [S.a. a]), but on closer inspection you will notice that there are only 21 different types of chromosomes. Thus, there is a pair of each type of chromosome and because of this, a rat's cells are said to be diploid (a word basically meaning having chromosomes in pairs) (Keeton 1973).

2.3 Mitosis

We know that a rat grows and is maintained through cell division. When a somatic cell (which refers to all cells in the body except for the reproductive cells) divides, each of the two resulting new daughter cells has exactly the same amount of chromosomes as the original cell (NZC 1994). But how is this achieved?

When a cell's nucleus divides, the genetic material is first duplicated and then distributed into each daughter cell. Before the parental cell divide, each strand of DNA contained within each chromosome, unwinds and is split into the two strands of DNA. Each strand is now a blueprint from which the other stand is rebuilt. After this process there are two copies of each chromosome which is equally distributed into each daughter cell upon cell division. The two daughter cells now contain exactly the same number and type of chromosomes as the parental cell (see figure 3) (Keeton 1973 & NZC 1994).

Mitosis is however only used to divide somatic cells, to allow the rat to grow and to replace old and worn-out cells (NZC 1994). To form the reproductive cells, a different process is used, namely meiosis.

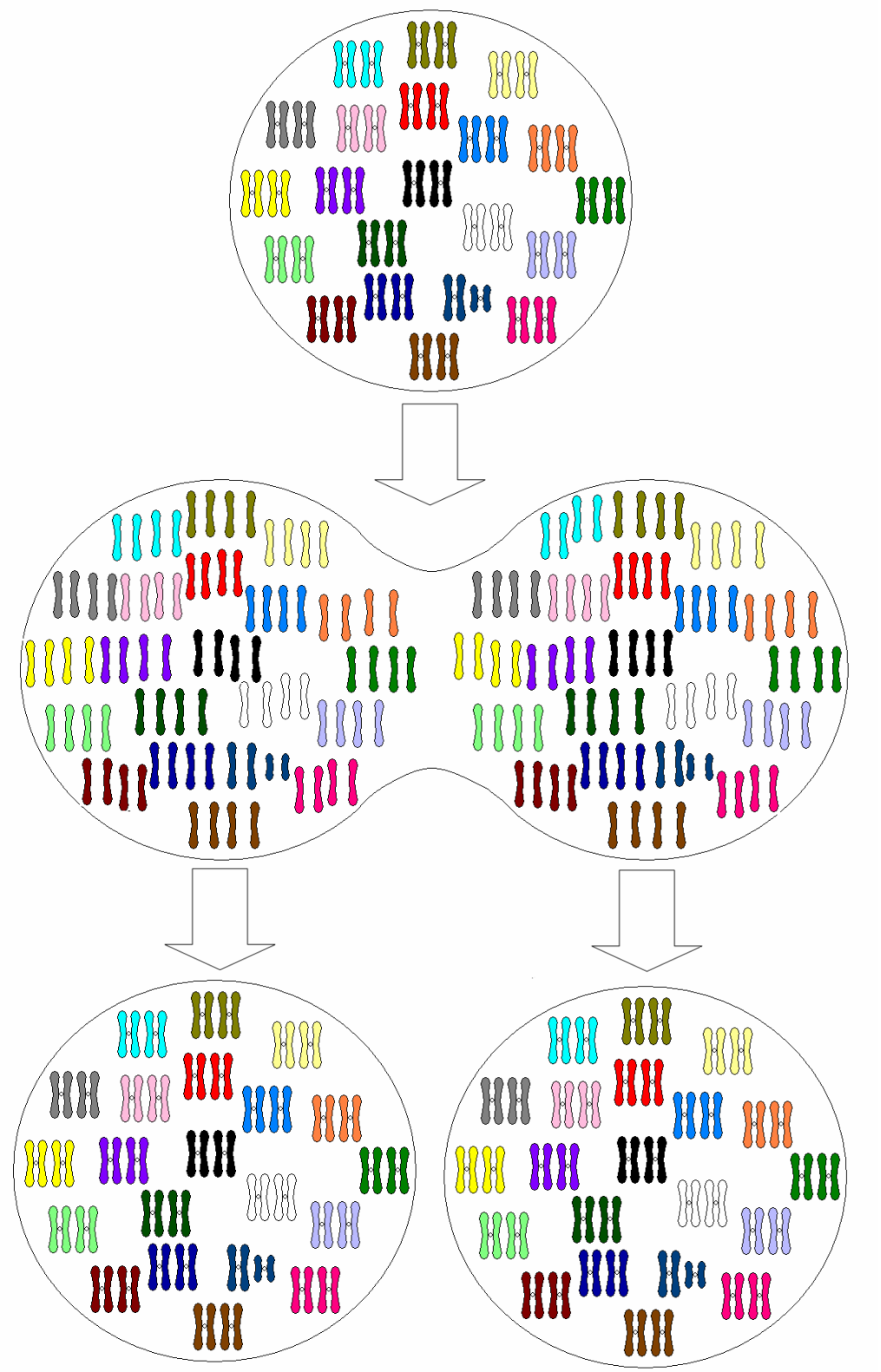


Figure 3: The process of mitosis

2.4 Meiosis

Earlier is mentioned that a rat has 42 chromosomes and that a rat's cells are diploid, thus having 21 pairs of each type of chromosome. We also know now, that during fertilization, a male's reproductive cell (or the sperm cell) unites with the female's reproductive cell (or the egg cell) to form the first cell (or the zygote) of the new baby rat. However, if the parent's reproductive cells (or germ cells (Grant 2006)) were formed through normal mitosis, each germ cell would have 42 chromosomes and upon uniting and the resulting zygote would have 84 chromosomes. However, this does not happen, as in reality each of the parent's germ cells only have half the normal amount of chromosomes. Thus, a germ cell only has one of each type of chromosome (21 chromosomes) instead of 2 of each type of chromosome. Such a cell, with only one of each type of chromosome, is said to be haploid. The germ cells (which are haploid) are also referred to as gametes and when male and the female gametes unite upon fertilization, the resulting zygote is again diploid (Keeton 1973 & NZC 1994).

Thus the process of meiosis is used to form haploid germ cells or gametes. Figure 4 shows what happens. Meiosis is a bit different from mitosis in that it occurs in 2 phases. In the first division phase, the 42 chromosomes are reduced to only one of each type of chromosome in the two daughter cells. Thus, at the end of phase one, each daughter cell has 21 chromosomes of only one type (the daughter cells are haploid). The second division phase occurs essentially in the same way as in mitosis to form four gametes. Here the chromosomes of each of the daughter cells from phase one divide in half. Each of these half chromosomes is now the blueprint from which the other half is again rebuilt. In the resulting two gametes from each daughter cell, the chromosomes are again complete, but still haploid (there is still 21 chromosomes of only one of each type) (Keeton 1973 & NZC 1994). Thus, now we can see that when the male and the female gamete unite, the resulting zygote has 42 chromosomes and not 48.

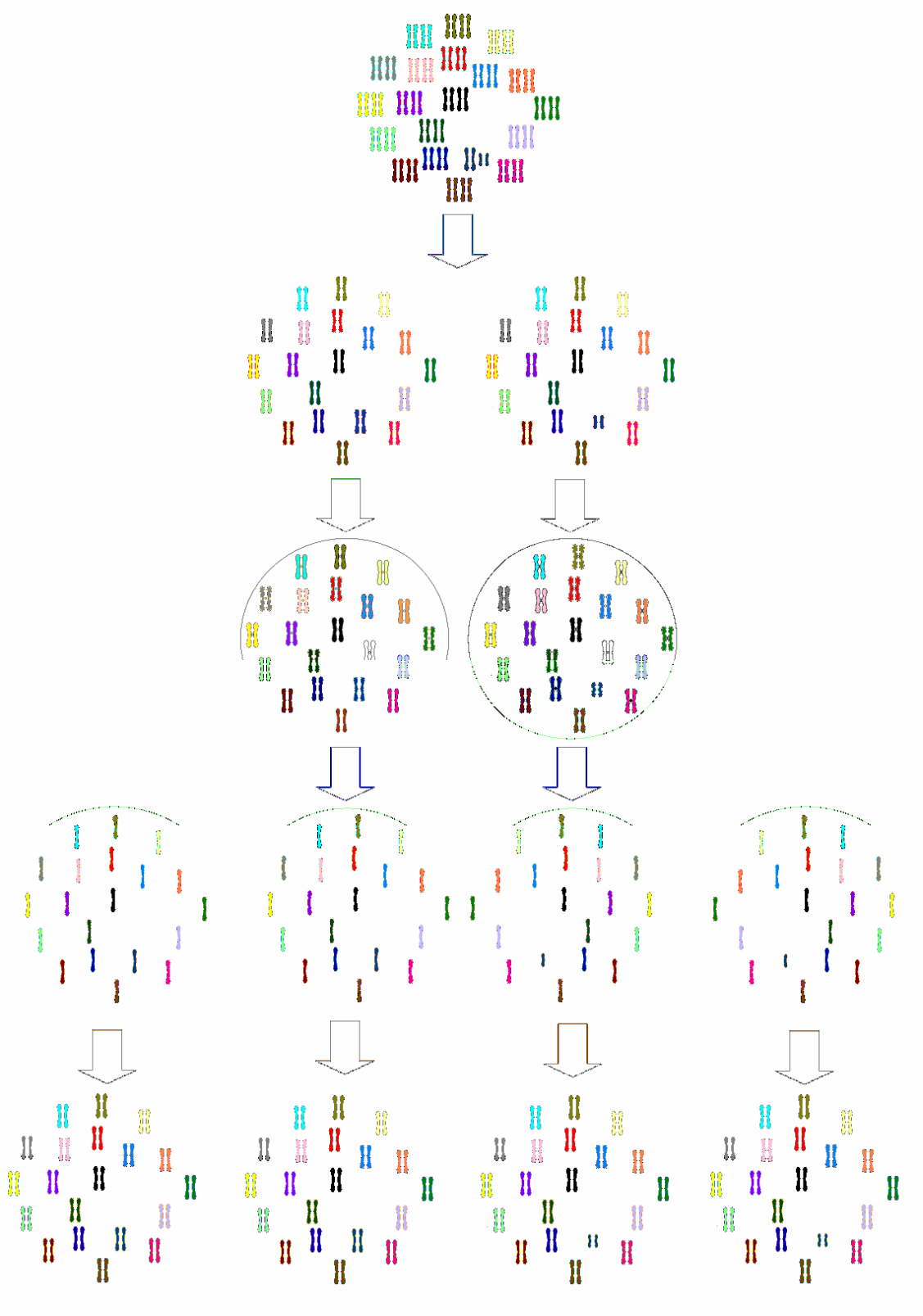


Figure 4: The process of meiosis

3 A closer look at the chromosomes

From now on we will use a more simplified method to illustrate a chromosome (shown in figure 5). This is to aid in explaining the following sections.

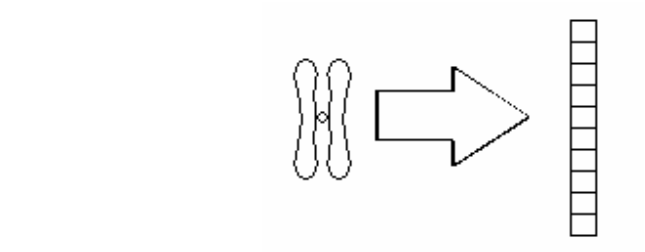


Figure 5: Simplified representation of a chromosome

3.1 Autosomal chromosomes

We already know that a rat has 42 chromosomes and that there are 21 different types of chromosomes. On closer inspection you will notice that with 20 of the pairs of chromosomes, each chromosome looks exactly like its corresponding partner. We call such chromosomes, homologous chromosomes (Keeton 1973). There are 20 pairs of homologous chromosomes in the rat, where in each pair; the two corresponding chromosomes are built exactly the same. These 20 pairs of chromosomes are known as the autosomal chromosomes (NZC 1994). There is another pair known as the sex chromosomes, but we will look at it below.

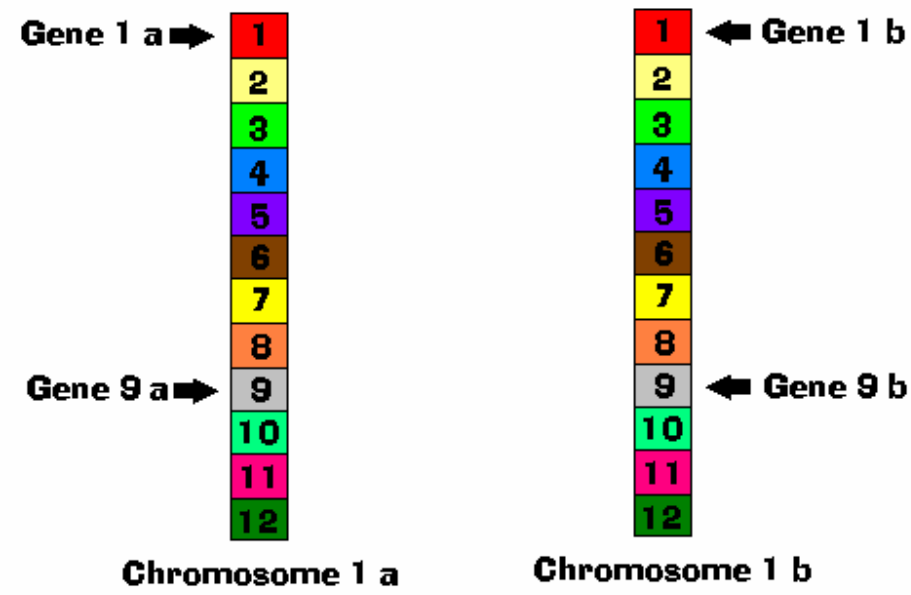


Figure 6: Placement of genes on chromosomal pair 1

When looking at a particular pair of autosomal chromosomes, you will notice that they are not only exactly the same length, but that a gene is also placed in exactly the same position as its counterpart on the corresponding chromosome (see figure 6).

But because there are always two copies of an autosomal chromosome pair, you will always need two copies of the same gene. The significance of this will be seen in section 4.

3.2 Sex chromosomes

The 21st pair of chromosomes or the sex chromosomes is a bit different from the autosomal chromosomes. There are two types of sex chromosomes, one known as the X chromosome (which has many genes located on it) and one known as the Y chromosome (which has only a few genes located on it) (Keeton 1973). Female rats have two copies of the X chromosome (see figure 7), thus with females the sex chromosome pair is also homologous. In male rats it is a different story. A male has one copy of the X chromosome (see figure 7) and one copy of the Y chromosome, thus his sex chromosome pair is not homologous.

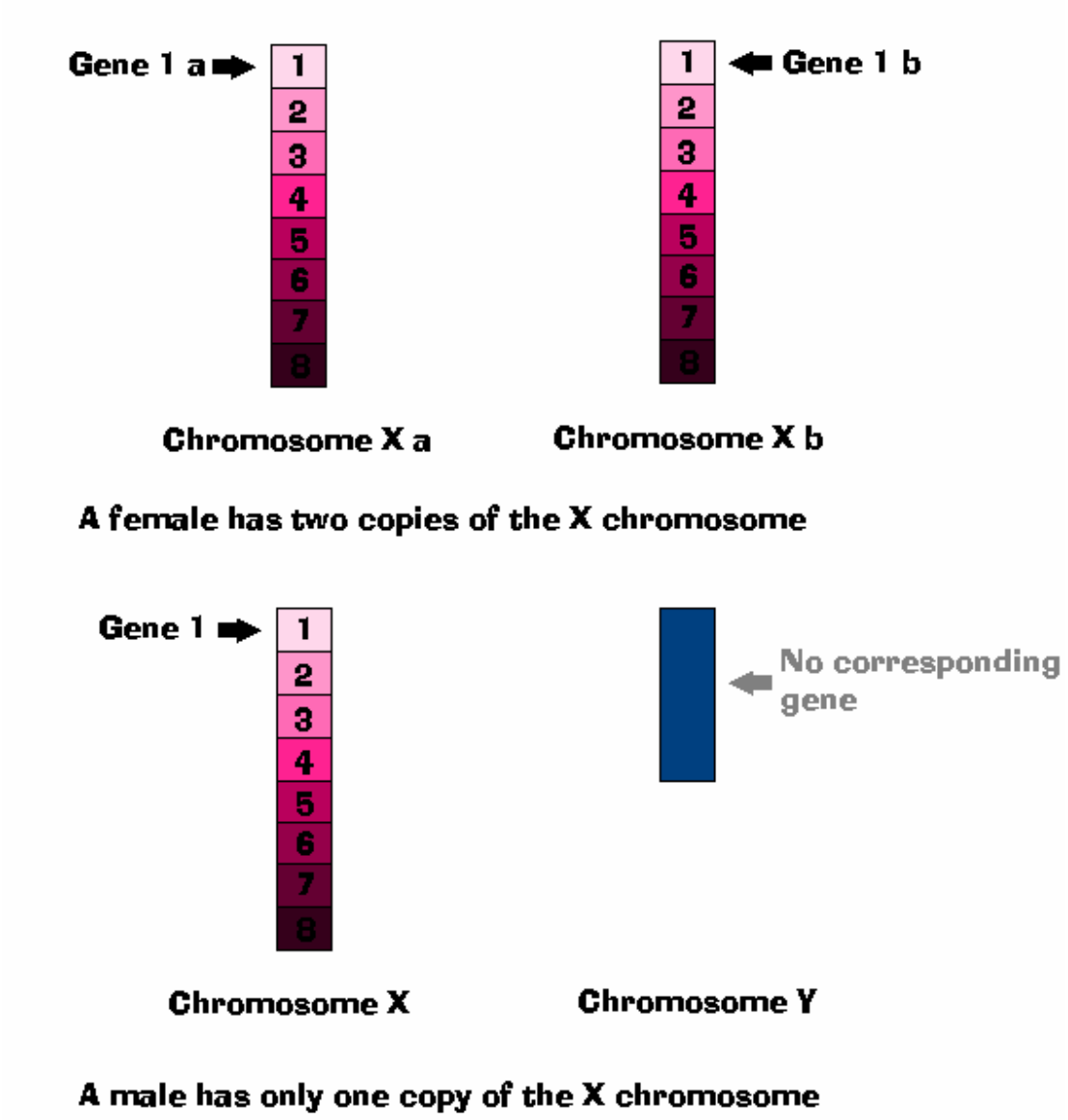


Figure 7: Placement of genes on the sex determining chromosomal pair

When considering genes on the X chromosome, a female always need two copies of a particular gene as above, but a male only need one copy as he only has one X chromosome. The Y chromosome is in most species usually very small and usually don't have any mutations on this chromosome. This is also the case with rats, and the only effect the sex chromosomes at the moment have for rats is determining a rat's sex, but we will come to this later.

4 Inheritance

During the years of 1856 to 1868 an Austrian monk, Gregor Mendel did experiments on garden peas and discovered that factors such as the colour of seedpods are inherited. From these experiments he determined his laws of inheritance, which is still largely used today (Keeton 1973).

4.1 Mutations

In section 1 we said the non-mutated version of the fancy rat is a standard ear agouti self. In genetic terms the non-mutated version of a specie is normally referred to as the wildtype. I also said there are many factors that determine the wildtype form of a fancy rat. If a fault occurs on one of these factors or genes, the gene is said to be mutated. The resulting effect of a mutated gene is called a mutation, e.g. for instance dumbo ears are a mutation of a factor that determines ear shape. The wildtype form of this factor is called standard ears.

4.2 Monohybrid inheritance

Monohybrid inheritance is the inheritance of a single factor or gene (Keeton 1973). When you cross an agouti rat with a black rat, all the babies will be agoutis. So what happened to the black? To explain this you will need to understand the laws which govern exactly how genes are inherited.

We can determine from the results of the above-mentioned cross that the agouti (or wildtype factor) is stronger than black (the mutated factor). In genetic terms we say agouti is dominant over black or that black is recessive to agouti. We also call the babies from this first cross the first filial generation[1], or abbreviated the F1 generation. If we now cross two of these F1 generation agouti babies together, we will get more agoutis and also some blacks in the F2 generation. This is because the recessive black factor is "hidden" in the F1 generation, but is now again expressed in the F2 generation. It is also said that the F1 agoutis carry the recessive mutated gene for black.

Although the parent agouti rat and the F1 generation agouti rats physically look the same, they have a different genetic makeup. The physical outward appearance (thus what you can see) is called the phenotype, while a rats genetic makeup is referred to as the genotype (Grant 2006).

Normally signs are used to express the above-mentioned crosses. There is also a specific way these signs should be written. For dominant genes a capital letter is used, and for agouti the letter **A** was chosen.

For recessive genes small caps are used, thus black would be symbolized by the letter **a** (Robinson 1965). However, you will need to write two letters when expressing the genotype of the parent rats as there are always two copies of each gene. Thus for the agouti parent we write A//A. The forward slashes are to indicate each gene of *one* pair of chromosomes, e.g. gene 1a on chromosome 1a ***//*** gene 1b on chromosome 1 b[2] (see figure 6) (NZC 1994).

For the black parent we write a//a and the cross of agouti × black is now represented as (A//A) × (a//a). As we explained in section 2.4, when meiosis occurs four haploid gametes are formed. The parental cell was diploid. Figure 8 is a simplified version of figure 4 and shows what happens in our cross during the process of meiosis.

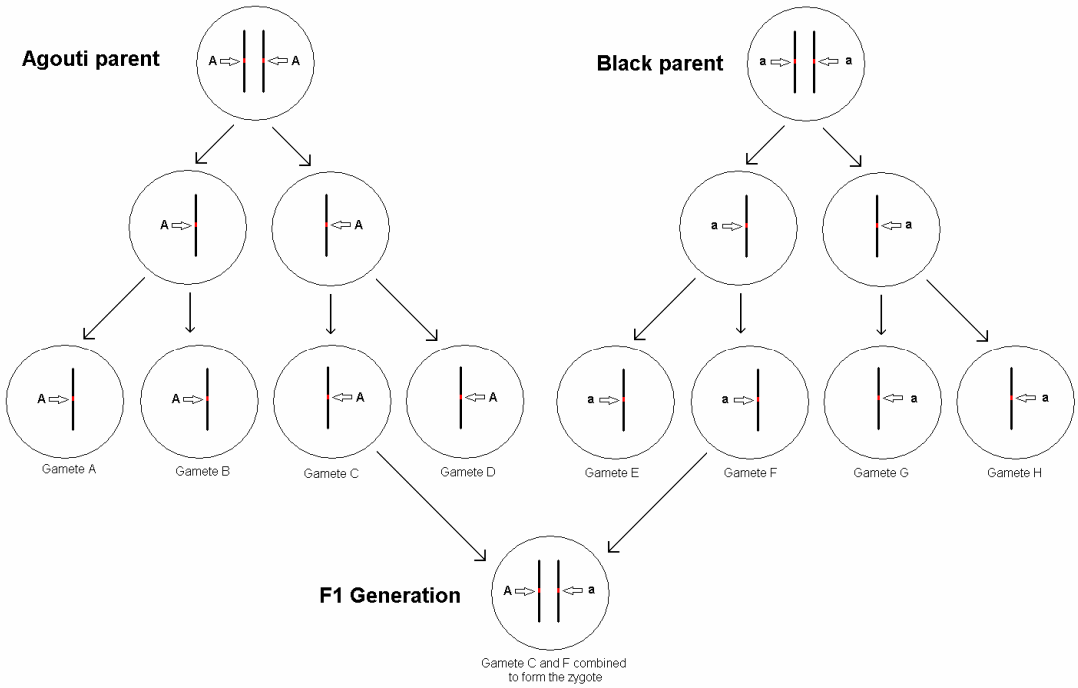


Figure 8: The process of meiosis during monohybrid inheritance represented by the agouti (A//A) X black (a//a) cross (only chromosome 3 shown).

Each parent rat's cell form four, haploid gametes with only one copy of each type of chromosome (in figure 8 only the chromosome on which the agouti factor is located is shown). Because of this we now write the gametes for each parent as such, the agouti parent has four A/ gametes and the black parent has four a/ gametes[2] (to avoid confusion, I will write the gametes in blue for this article and bold when referring to a gene's symbol).

As can be seen from figure 8, all the gametes are numbered. Gamete A, B, C and D are used for the agouti parent and gametes E, F, G and H for the black parent.

Any combination of two of these eight gametes can combine to form the zygote: Gamete A from the agouti parent can combine with Gamete E or F or H or G of the black parent. Thus, any of the following combinations is possible:

Agouti parent's gametes	Black parent's gametes	Resulting F1 zygote can be formed by:
Gamete A	Gamete E	Gamete A & E
Gamete A	Gamete F	Gamete A & F
Gamete A	Gamete G	Gamete A & G
Gamete A	Gamete H	Gamete A & H
Gamete B	Gamete E	Gamete B & E
Gamete B	Gamete F	Gamete B & F
Gamete B	Gamete G	Gamete B & G
Gamete B	Gamete H	Gamete B & H
Gamete C	Gamete E	Gamete C & E
Gamete C	Gamete F	Gamete C & F
Gamete C	Gamete G	Gamete C & G
Gamete C	Gamete H	Gamete C & H
Gamete D	Gamete E	Gamete D & E
Gamete D	Gamete F	Gamete D & F
Gamete D	Gamete G	Gamete D & G
Gamete D	Gamete H	Gamete D & H

You can now see that there are 16 different combinations possible, but the question is, which gamete combination will the zygote be? We cannot determine this. That is why working out genetic combinations is sometimes like throwing dice, you never know on which side the dice is going to land (NZC 1994). However, for our cross of the agouti and the black rat, which combination it will be, does not matter, as all the gametes are the same and will all give the same result. But later when we work with combinations where not all the gametes are the same for a single parent, then it does matter which gamete combines with which.

When the gametes of the parents combine, A/ for the agouti parent and a/ for the black parent, we now have a F1 generation with the genotype A//a. Thus, (A//A) $\times$ (a//a) = (A//a). Yet, there is even an easier method to show what happens in figure 8. Reginalt Punnett developed a method to represent genetic crosses. This is known as the Punnett square method (Wikipedia [S.a. b]). This is how it works; you draw a grit as shown here:

Father's gametes

Mother's gametes	Gamete A	Gamete B	Gamete C	Gamete D
Gamete E	A & E	B & E	C & E	D & E
Gamete F	A & F	B & F	C & F	D & F
Gamete G	A & G	B & G	C & G	D & G
Gamete H	A & H	B & H	C & H	D & H

The father's gametes are filled in into the top row (shown in blue) and the mother's gametes in the first column to the left (shown in pink) [you can also do it the other way round, e.g. mother in top row, it does not really matter]. The combinations of the parent's gametes are now filled into the slots (shown in white); these will be the possible combinations of the offspring.

Let us do it for the agouti × black cross:

		Agouti parent			
		A/	A/	A/	A/
Black parent	a/	A//a	A//a	A//a	A//a
	a/	A//a	A//a	A//a	A//a
	a/	A//a	A//a	A//a	A//a
	a/	A//a	A//a	A//a	A//a

We can see that all of our F1 generation will be agoutis which carry black (A//a). Normally we do not write each and every gamete, *if* they are identical. Thus, our cross represented above can also be demonstrated as such:

		Agouti parent
		A/
Black parent	a/	A//a

If we now cross two of the F1 generation offspring together, we will get an interesting result.

		F1 agouti parent	
		A/	a/
F1 agouti parent	A/	A//A	A//a
	a/	A//a	a//a

1/4 of the offspring will be agouti's (A//A), 1/2 of the offspring will be agouti's which carry black (A//a) and 1/4 will be blacks. To introduce more genetic terms, the A//A agouti offspring is said to be homozygous agoutis. Mutations are homozygous when two corresponding genes are the same. The agouti's which carry black (A//a) are said to be heterozygous agoutis. Heterozygous mutations have two corresponding genes which are *not* the same (Keeton 1973). Thus although all the agoutis in this litter look the same (have the same phenotype) they differ genetically (have a different genotype).

4.2.1 Autosomal recessive factors

Autosomal recessive factors are basically genes located on any of the 20 autosomal chromosomes of the rat (see section 3.1). We know that there are always two chromosomes in an autosomal pair and therefore we always need to write two copies of a gene (see figure 6). Recessive mutations are those mutations where the wildtype factor is dominant over the corresponding mutated factor. The black we discussed above is such a mutation. Black (or more correctly named non-agouti) is the mutated form of the agouti factor. Normally the agouti factor or gene is responsible for the banding effect of individual hairs (alternation between brown-black and red-yellow pigment production in the hair) and therefore the resulting colour of the agouti coloured rat. The non-agouti factor is a mutation which occurred on this gene and resulted in that process behind the banding of individual hairs cannot function normally (no alternation, only brown-black pigment produced continuously), resulting in a single coloured hair and thus giving a rat with a black colour (Rat Biology 2004b). However, because this non-agouti mutation is recessive, two copies of the mutated gene is needed to give the phenotype black. When this gene is not mutated the rat's phenotype is agouti. Thus A//A is an agouti, but also A//a because the **A** wildtype gene is dominant over **a**, resulting in that **a** cannot express phenotypically. a//a is, however, a black rat as there is not dominant **A** present to prevent expression of the recessive non-agouti mutation.

Non-agouti is not the only autosomal recessive mutation in our fancy rats. There are many other autosomal recessive mutations. Pink eyed dilution for instance is another autosomal recessive mutation which changed the wildtype factor responsible for controlling black-brown colouring in the rat, but leaving yellow-red colouring unaffected. When this gene is mutated only the yellow-red colouring is produced normally while black-brown is reduced, resulting in an amber rat (Rat Biology 2004b). We use the symbol **p** for pink eyed dilution, thus the wildtype for this gene is P//P giving an agouti rat. P//p also gives an agouti phenotype while when you have two copies of the mutated **p** gene you get an amber rat (p//p), *if* there are no other mutated genes. We normally write amber as A//A p//p, indicating that the agouti gene is also non-mutated. In section 4.3 we will go into more detail on this.

4.2.2 Autosomal dominant factors

Autosomal dominant factors are basically the same as autosomal recessive factors, except that with autosomal dominant factors the mutated factor is dominant over the wildtype factor. We will use the well known autosomal dominant rex mutation, which results in a curly coat instead of the normal standard straight coat, as an example.

Because our mutated rex gene is dominant over the wildtype standard coat, we write the rex gene with initial capital letters, **Re** (Rat biology 2004a). The wildtype of this gene is now written as **re**, indicating that it is recessive over the mutated **Re**.

Thus a Re//re rat has a rex coat and not a wildtype standard coat as expected. A standard coat would be re//re. All autosomal dominant mutations can be phenotypically expressed if there is only one copy of the mutated gene. But often when there are two copies of the mutated gene a slightly different phenotype results. Re//Re rats are called double rexes and phenotypically seem semi hairless as the double dose of the rex gene now results in some hair loss leaving patches of almost bare skin (Rat biology 2004a). Some autosomal dominant factors result in a lethal phenotype (see section 4.10), while others give the same phenotype as when expressed in a single dose.

One important thing to note with autosomal dominant mutations is that it CANNOT be carried. For instance, you either have a rex or you don't, any other non-rex rat cannot carry the mutated rex gene or give rex offspring if mated to a non-rex[3].

4.2.3 Sex linked factors

Autosomal factors are linked to any of the 20 pairs of autosomal chromosomes of the rat. Sex linked factors, as the name suggest, is linked to the 21st pair of sex chromosomes. Before we go into a discussion on mutations on sex chromosomes we are first going to look at the other main function of the sex chromosomes, which is to determine a rat's sex. In section 3.2 we said a female rat has two copies of the X chromosome (XX) and the male only one (XY). When any male and female rat mates, you always get ½ female and a ½ male offspring:

		Male	
		X	Y
Female	X	XX	XY
	X	XX	XY

There is no way to change this ratio, you always have a 50% chance of female and a 50% chance of males. However, when mutations occur on the sex chromosomes, the resulting phenotype of the mutation is also linked to the animal's sex. Sadly, we do not yet have any fancy rat varieties which are caused by mutations linked to the sex chromosomes. In other species there are many sex linked genes, such as in hamsters you get a tortoiseshell variety (River road hamstery 2000). In future we also hope to get this variety in rats, thus I am still going to explain sex linked inheritance even though at this point we do not have any sex linked fancy rat varieties.

For this section I have chosen the domestic cat as our model to explain sex linked inheritance, as the cat varieties we will target here are very common in our pet cat population in South Africa.

Everyone has probably heard of ginger or orange coloured cats (see figure 9) (or more appropriately named the red tabbies (McHoy 1987)) and also of tortoiseshell coloured cats (see figure 10).

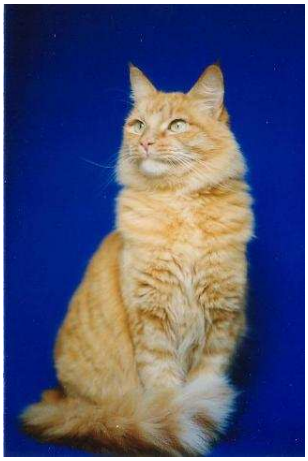


Figure 9: A ginger or red tabby coloured female cat

Figure 10: A tortoiseshell coloured female cat

These two colour varieties are caused by a single dominant gene located on the X chromosome of the cat. The gene is named orange and the symbol they use is **O** (Giant book of the cat 2002). Because male cats (and rats) only have one X chromosome (remember section 3.2) we cannot write the genotype as we did above. To avoid confusion and because the sex of the animal also plays an important role in this case, we are now going to write the chromosome (X or Y in this case) next to the symbol of the gene. A male red tabby cat has the genotype: XO//Y whilst a female tortoiseshell cat has the genotype: XO//Xo, if you mate these two cats together your will get the following offspring:

		Red tabby male	
		XO/	Y/
Tortoiseshell female	XO/	XO//XO	XO//Y
	Xo/	XO//Xo	Xo//Y

¼ of the kittens will be female tortoiseshell (XO//Xo)
¼ will be female red tabby (XO//XO)
¼ will be male brown tabby (Xo//Y)
and ¼ will be male red tabby (XO//Y) (Giant book of the cat 2002)

From the above cross you can see that only female cats can be tortoiseshell[4] as only females have the necessary two copies of the X chromosome to cause this mutation. It is commonly believed that only male cats can be red tabbies, but in reality females can also be red tabbies. Female red tabbies are just a bit more scarce than the males because both X chromosomes must have the dominant **O** gene giving a smaller chance that the right combination will be achieved (tortoiseshell female $\times$ red tabby male) to bring two X chromosomes together having the dominant **O** gene. On the other hand, male red tabbies are common because they only have one X chromosome and the Y chromosome has no influence on this gene (it is a different kind of chromosome) (Giant book of the cat 2002).

So, what about the Y chromosome, can mutations occur on it? The answer is yes there can be mutations on the Y chromosome, but as the Y chromosome is so small and therefore has so few genes on it, that the chances are very small for a mutation to occur on this chromosome.

4.2.4 Multiple alleles

From our discussion above, we saw that an individual gene may have two forms or alleles, namely the original wildtype form and a mutated form. But sometimes a particular gene has more than two alleles (or multiple alleles). This happens when a gene gets mutated more than once (Keeton 1973). For instance, someone in America might have a new rat variety as a result of a mutation on a particular gene, then, say someone in Europe also has a new variety where another mutation occurred on the same gene as the rat in America. Now this gene has got three alleles.

In the rat fancy there are two genes which have multiple alleles. One is the factor controlling the presence or absence of colour in the rat (the colour locus or C-locus[5]) (Rat Biology 2004b) and the other one controls the amount of a certain type of white spotting in the rat (the hooded locus or H-locus) (Rat Biology 2004b). I will use the C-locus to explain this section.

The wildtype form of this C-locus is symbolized as **C** (Robinson 1965), giving a rat which is fully pigmented. A mutation occurred that causes a rat with no colour pigment at all, it is known as the albino rat or in the fancy as pink eyed white. However, this albino mutation is recessive to wildtype and therefore is symbolized as **c**. Thus to summarize: C//C is a fully pigmented rat, C//c is also a fully pigmented rat but carries the non-pigment mutation, albinism (Robinson 1965). c//c is a non-pigmented rat (phenotipically an albino).

Yet, this is not all; later the C gene mutated again, now giving another mutation that does not fully remove all the pigment (an acromelanistic allele giving our siamese rats[6]) (Rat Biology 2004b). Now how do we symbolize this mutation? Small caps **c** is already taken for the non-pigment mutation (albinism) which is recessive to wildtype.

This new mutation is also recessive to wildtype, thus must also be written in small caps. We must also still use the letter c (another letter will indicate another gene), thus now we still write c but put in a superscript h to it to indicate that it is not the albinism **c**, but another recessive mutation to wildtype. We now write $\mathbf{c^h}$ for this new mutation. C//C still indicates a full coloured rat (same as above) and $C//c^h$ is also a full coloured rat carrying the siamese gene and $c^h//c^h$ is a siamese rat.

Yet you also now get a $c^h//c$ genotype and the resulting colour will now depend on which mutation is dominant over the other one. Say if $\mathbf{c^h}$ is dominant over **c**, you would expect a siamese rat or if **c** is dominant over $\mathbf{c^h}$ you would expect an albino rat. However, this is not the case here, neither of these two mutated genes is fully dominant over the other and therefore $c^h//c$ is actually an intermediate form, the himalayan rat. This brings us to the next section, intermediate inheritance.

4.2.5 Intermediate inheritance

Intermediate inheritance occurs when two alleles on the same gene are neither recessive nor dominant two each other (thus, they are co-dominant to each other). In the plant kingdom, certain species, such as snapdragons, express intermediate inheritance. When a red snapdragon is crossed with a white snapdragon, pink offspring is obtained. This is because neither the allele for red ($\mathbf{C^r}$) nor the allele for white ($\mathbf{C^w}$) is dominant over the other, therefore a phenotype results which express effects of both alleles (Keeton 1973).

White snapdragon \ Red snapdragon	$C^r/$	$C^r/$
$C^w/$	$C^w//C^r$	$C^w//C^r$
$C^w/$	$C^w//C^r$	$C^w//C^r$

Thus a red snapdragon ($C^r//C^r$) crossed with a white snapdragon ($C^w//C^w$) gives all pink snapdragons ($C^w//C^r$).

The same happens with a cross between homozygous albino (c//c) and siamese ($c^h//c^h$) fancy rats. The heterozygous himalayan ($c^h//c$) offspring have effects of both the albino and the siamese parents, thus its phenotype is intermediate to the two phenotypes of the parents.

Albino \ Siamese	$c^h/$	$c^h/$
$c/$	$c^h//c$	$c^h//c$
$c/$	$c^h//c$	$c^h//c$

4.3 Di-hybrid inheritance

Di-hybrid inheritance is the inheritance of two genes (Keeton 1973). We've seen what happens if you cross rats where only a single gene is mutated, but what if you cross two rats each mutated by a different gene? We know that a black rat is written as a//a and an amber rat as p//p, but when we cross these two colours with each other (see figure 11), we must write a//a P//P for the black and A//A p//p for the amber because we must represent both genes in both parents no matter if they are mutated or not.

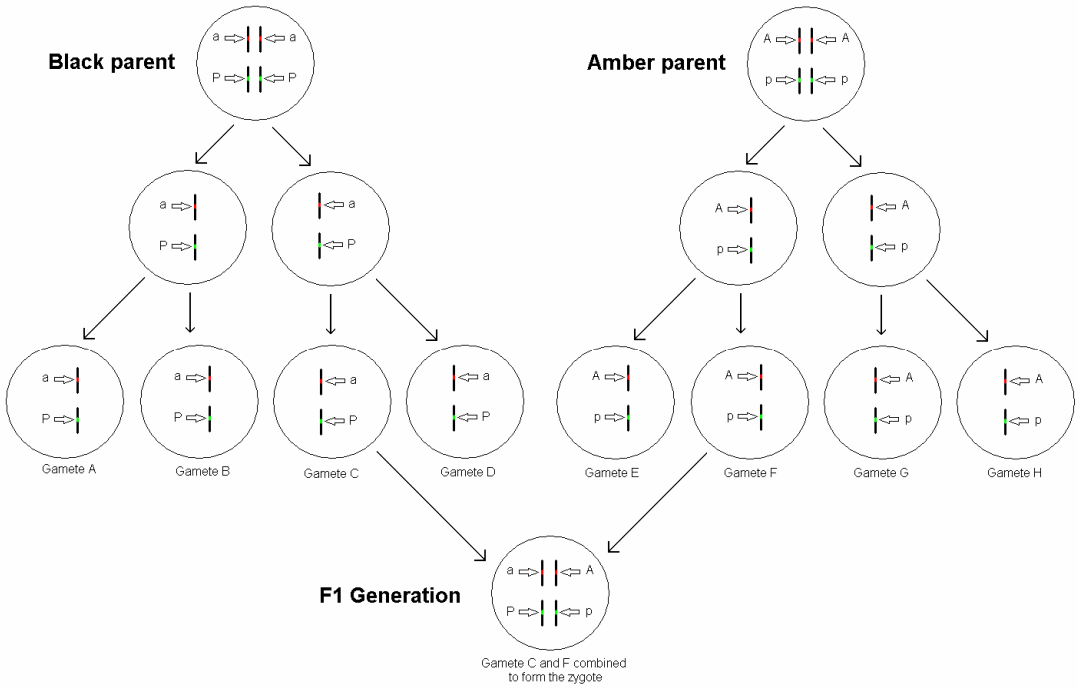


Figure 11: The process of meiosis during dihybrid inheritance represented by the amber (A//A p//p) X black (a//a P//P) cross (only chromosome 1 and 3 shown).

The black rat will now have four a/ P/ gametes, while the amber rat will have four A/ p/ gametes. The F1 offspring have now a A//a P//p genotype. The a/ of the black parent is always written together with the A/ of the amber parent, because this is one gene, the same happens with the P/ and p/ gametes. The F1 offspring will be agouti because in both mutations the mutated factor is recessive. Thus, the non-mutated factor dominates the mutated factor and therefore suppresses it. If we look at the F1 offspring's genotype, we can see that **A** is dominant over **a**, meaning that the babies will have agouti ticking and also that **P** dominates over **p** meaning that the baby will also not be diluted, thus the babies will be agoutis.

The black (a//a P//P) × amber (A//A p//p) cross is represented as follows:

		Amber parent
		A/ p/
Black parent	a/ P/	A//a P//p

If two of the F1 offspring are crossed with each other, the resulting litter will be agouti, black, amber and champagne in a 9 : 3 : 3 : 1 ratio (or in other words 56% or 9/16 chance for agouti; 19% or 3/16 chance for black;19% or 3/16 chance for amber and 6% or 1/16 chance for champagne[7]). Each of the F1 parents will have the following gametes: A/ P/; A/ p/; a/ P/ and a/ p/.

An agouti F1 male crossed with an agouti F1 female is represented as follows:

		F1 agouti parent			
		A/ P/	A/ p/	a/ P/	a/ p/
F1 agouti parent	A/ P/	A//A P//P agouti	A//A P//p agouti	A//a P//P agouti	A//a P//p agouti
	A/ p/	A//A P//p agouti	A//A p//p amber	A//a P//p agouti	A//a p//p amber
	a/ P/	A//a P//P agouti	A//a P//p agouti	a//a P//P black	a//a P//p black
	a/ p/	A//a P//p agouti	A//a p//p amber	a//a P//p black	a//a p//p champagne

In section 4.2 we said that the genotypes which are filled into the slots of a Punnett square are the *possible* combination for the offspring. Because we do not know which gametes will combine and which zygotes will grow to form a baby, it does not mean that the above ratio of colours will come out exactly as stated above. For instance, you might not have a champagne baby in that particular litter or you might get more of a particular colour than expected. Now if you do the above cross, say 50 times and count all the different coloured babies of all the litters you will probably get the ratio described above.

It should be obvious that how smaller a percentage or ratio become for a particular colour, the less likely it will be that such a baby will be produced in a particular litter. That is also why some colours are so rare.

4.4 Tri-hybrid inheritance

Tri-hybrid inheritance is the inheritance of 3 genes. One also gets tetra-hybrid inheritance (4 genes), penta-hybrid inheritance (5 genes), etc, etc. But the more genes involved the smaller the ratios become for each colour. If you want to breed a russian silver point siamese, but you only have a seal point siamese (a//a c^h//c^h D//D G//G) and a russian silver (a//a C//C d//d g//g) to start with, you will only have a 1/64 chance to breed this colour, a very small chance indeed!

Seal point siamese (a/a c^h//c^h D//D G//G) × Russian silver (a//a C//C d//d g//g):

Seal point siamese

Russian silver	c^h/ D/ G/
C/ d/ g/	C//c^h D//d G//g

F1 parent (C//c^h D//d G//g) × F1 parent (C//c^h D//d G//g):

F 1 black parent

F1 black parent	C/ D/ G/	C/ D/ g/	C/ d/ G/	C/ d/ g/	c^h/ D/ G/	c^h/ D/ g/	c^h/ d/ G/	c^h/ d/ g/
C/ D/ G/	C//C D//D G//G black	C//C D//D G//g black	C//C D//d G//G black	C//C D//d G//g black	C//c^h D//D G//G black	C//c^h D//D G//g black	C//c^h D//d G//G black	C//c^h D//d G//g black
C/ D/ g/	C//C D//D G//g black	C//C D//D g//g blue	C//C D//d G//g black	C//C D//d g//g blue	C//c^h D//D G//g black	C//c^h D//D g//g blue	C//c^h D//d G//g black	C//c^h D//d g//g blue
C/ d/ G/	C//C D//d G//G black	C//C D//d G//g black	C//C d//d G//G Russian blue	C//C d//d G//g Russian blue	C//c^h D//d G//G black	C//c^h D//d G//g black	C//c^h d//d G//G Russian blue	C//c^h d//d G//g Russian blue
C/ d/ g/	C//C D//d G//g black	C//C D//d g//g blue	C//C d//d G//g Russian blue	C//C d//d g//g Russian silver	C//c^h D//d G//g black	C//c^h D//d g//g blue	C//c^h d//d G//g Russian blue	C//c^h d//d g//g Russian silver
c^h/ D/ G/	C//c^h D//D G//G black	C//c^h D//D G//g black	C//c^h D//d G//G black	C//c^h D//d G//g black	c^h//c^h D//D G//G Seal point siamese	c^h//c^h D//D G//g Seal point siamese	c^h//c^h D//d G//G Seal point siamese	c^h//c^h D//d G//g Seal point siamese
c^h/ D/ g/	C//c^h D//D G//g black	C//c^h D//D g//g blue	C//c^h D//d G//g black	C//c^h D//d g//g blue	c^h//c^h D//D G//g Seal point siamese	c^h//c^h D//D g//g Blue point siamese	c^h//c^h D//d G//g Seal point siamese	c^h//c^h D//d g//g Blue point siamese

c^h/ d/ G/	C//c^h D//d G//G black	C//c^h D//d G//g black	C//c^h d//d G//G Russian blue	C//c^h d//d G//g Russian blue	c^h//c^h D//d G//G Seal point siamese	c^h//c^h D//d G//g Seal point siamese	c^h//c^h d//d G//G Russian blue point siamese	c^h//c^h d//d G//g Russian blue point siamese
c^h/ d/ g/	C//c^h D//d G//g black	C//c^h D//d g//g blue	C//c^h d//d G//g Russian blue	C//c^h d//d g//g Russian silver	c^h//c^h D//d G//g Seal point siamese	c^h//c^h D//d g//g Blue point siamese	c^h//c^h d//d G//g Russian blue point siamese	c^h//c^h d//d g//g Russian silver point siamese

Note that although the original seal point siamese was actually a result of di-hybrid inheritance (both mutated **a** and **c^h** present) and the Russian silver of tri-hydrid inheritance (mutated a, d and g present), we did not write mutated a in the equations above, as both parents have it. If both parents are mutated for the same factor one can leave it out of the equation, but one must just then remember that all the offspring is also mutated for that factor plus any other factors concerned.

4.5 Gene interactions

Sometimes di-hybrid crosses do not give the expected 9 : 3 : 3 : 1 ratio, but a 9 : 3 : 4 ratio, this is because some genes give the same phenotypic effect on different genetic backgrounds (Keeton 1973). For instance, if you cross a chocolate rat with an albino rat (or better known as pink eyed white in the fancy), the F1 offspring will be black as expected. But when you cross two of these F1 offspring with each other you do not get the expected ratio of 9 black; 3 chocolate; 3 PEW and 1 “white-chocolate”, but a ratio of 9 black; 3 chocolate and 4 PEW. This is because the recessive **c** gene expresses the same phenotype on both the black (a//a B//B c//c) and the chocolate (a//a b//b c//c) background. The reason for this is that the **c** gene takes away all the colour pigment from the coat resulting in a rat which looks white with pink eyes. Thus the **b** gene cannot show its effect when combined with **c** as there is no pigment present for the **b** gene to dilute. The effects of the recessive b//b combination is hidden and in genetic terms it is said that the **c** gene is epistatic (because it hides the effects of other genes, they are there, they just cannot be expressed phenotypically).

Chocolate (a//a b//b C//C) × PEW (a//a B//B c//c)

		Chocolate
		b/ C/
PEW	B/ c/	B//b C//c

F1 black parent

F1 black parent	B/ C/	B/ c/	b/ C/	b/ c/
B/ C/	B//B C//C Black	B//B C//c Black	B//b C//C Black	B//b C//c Black
B/ c/	B//B C//c Black	B//B c//c PEW	B//b C//c Black	B//b c//c PEW
b/ C/	B//b C//C Black	B//b C//c Black	b//b C//C Chocolate	b//b C//c Chocolate
b/ c/	B//b C//c Black	B//b c//c PEW	b//b C//c Chocolate	b//b c//c PEW

4.6 Linked factors

Linked factors are when two genes are located on the same chromosome (NZC 1994). In all the above examples where we discussed varieties caused by two or more mutated genes (such as in di-hybrid or tri-hybrid inheritance), the different genes were located on different chromosomes. But what happens when the genes are located on the same chromosome?

Both the pink eyed dilution locus and the colour-locus are located on chromosome one. Many attempts have been made in the past to breed an amber point siamese, but no-one has ever succeeded. On the other hand it is easy to breed a blue point siamese, but why can't we breed an amber point siamese? This is because pink eyed dilution and the himalayan allele are linked genes.

The first step in breeding amber point siamese would be to cross an agouti point siamese (A//A c^h//c^h P//P) with an amber rat (A//A C//C p//p). The F1 generation would all be agoutis as expected and the next logical step would be to cross two F1 offspring together. One expect a ratio of 9 : 3 : 3 : 1 agouti, seal point siamese, amber and amber point siamese (just like the cross discussed in section 4.3 to breed a champagne rat). Yet this does not happen, you get a ratio of 2 : 1 : 1 of agouti, amber and seal point siamese.

In section 4.2 it was shown how genetic formulas are written. There we said that for each gene we write for instance gene 1a on chromosome 1a ***//*** gene 1b on chromosome 1 b and again gene 2a on chromosome 2a ***//*** gene 2b on chromosome 2 b (see figure 6). This was when you considered only one gene on each chromosome, but when there is more than one gene on the same chromosome you write for instance gene 1a**,** gene 9a on chromosome 1a ***//*** gene 1b**,** gene 9b on chromosome 1 b (see figure 6) (NZC 1994). Thus instead of writing C//C p//p for amber (as this will indicate that **C** and **p** are located on two different chromosomes), we have to write A//A C,p//C,p for the amber and A//A c^h,P//c^h,P for the agouti point siamese. The amber parent will have four A/ C,p/ gametes and the agouti point siamese will have four A/ c^h,P/ gametes.

The cross between amber and agouti point siamese will look like this:

Agouti point siamese \ Amber	A/ C,p/
A/ c^h,P/	A//A C,p//c^h,P agouti

The offspring will all be agoutis as **C** still dominates **c^h** and **P** still still dominates **p**. If you now cross two of these F1 agouti offspring with each other, each F1 agouti will have two A/ C,p/ and two A/ c^h,P/ gametes. The cross is represented as follows:

F1 agouti parent \ F1 agouti parent	A/ C,p/	A/ c^h,P/
A/ C,p/	A//A C,p//C,p Amber	A//A C,p//c^hP Agouti
A/ c^h,P/	A//A C,p//c^h,P Agouti	A//A c^h,P //c^h,P Agouti point siamese

If you look closer at all the possible crosses above, you will notice that it is impossible to get an amber point siamese. Yet sometimes something strange happens in nature which allows mutations to occur that are caused by two genes on the same chromosome. This phenomenon is known as crossing over.

4.7 Crossing-over

The process of meiosis we discussed back in section 2.4 is actually very simplified. In reality it's a much more complicated process with many stages. At one of these stages the chromosomes appear as long thin threads, but sometimes one of these threads break. Usually the threadlike-chromosome quickly grows back together again, but rarely it happens that *both* chromosomes in a certain chromosomal pair breaks and the wrong parts grows back together again (see figure 12) (NZC 1994).

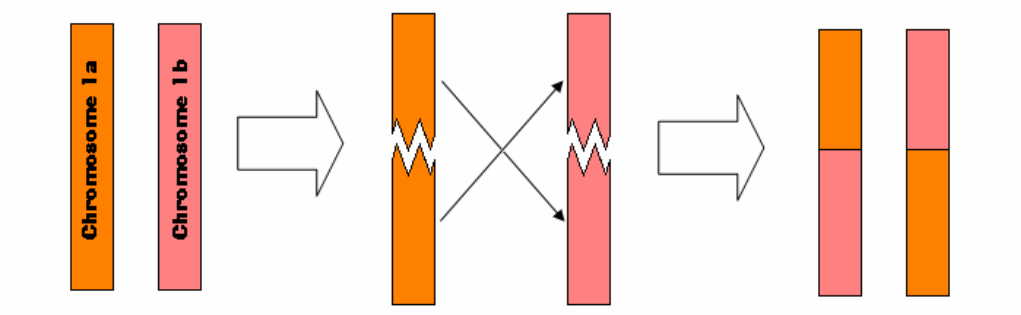


Figure 12: Crossing-over of chromosome 1

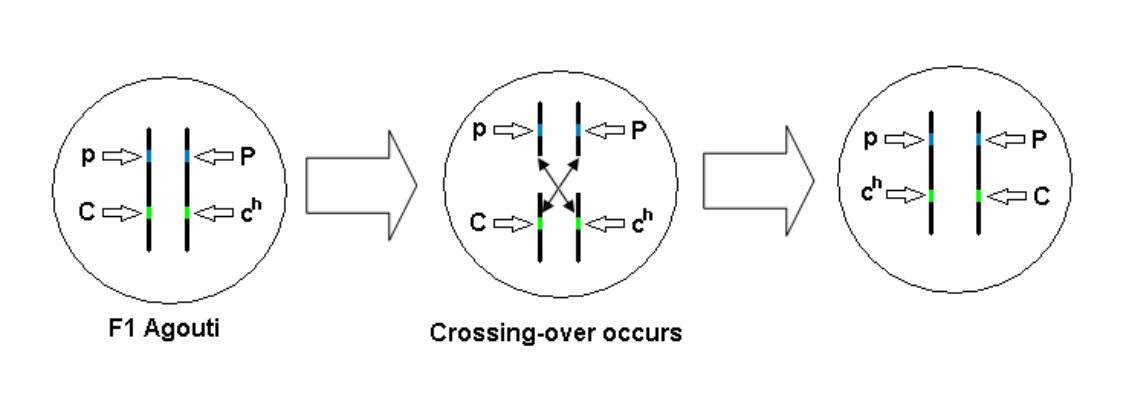


Figure 13: Crossing over occurs for a F1 agouti

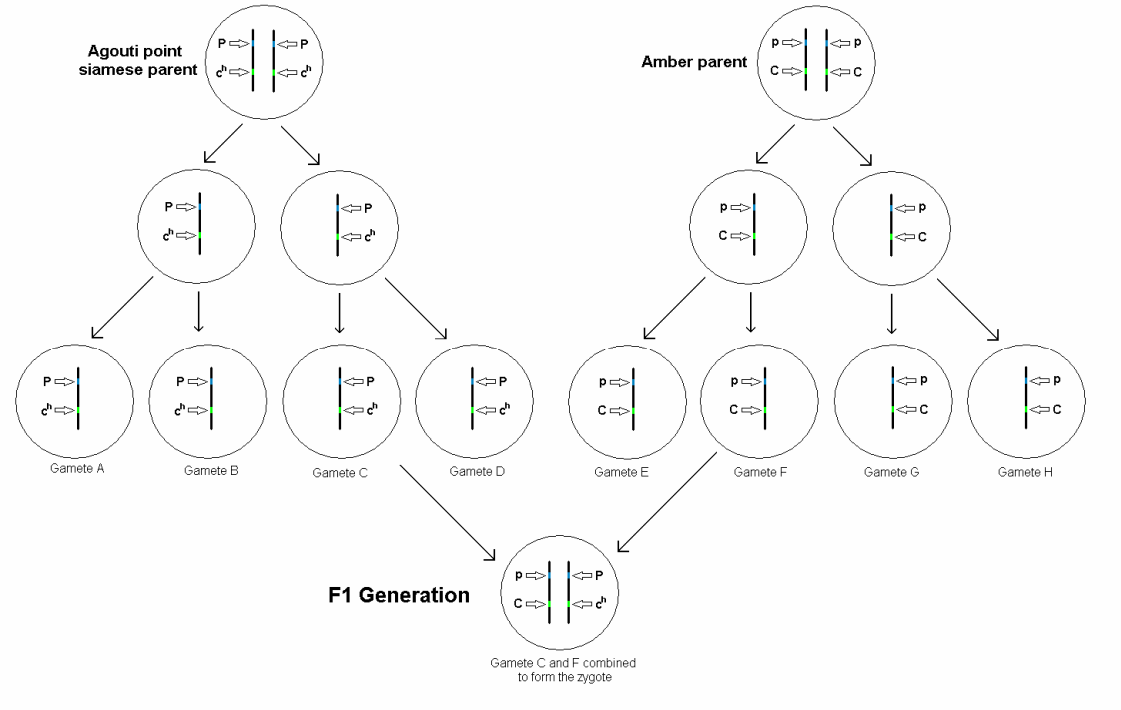


Figure 14: The process of meiosis without crossing over occurring for the amber × agouti point siamese cross

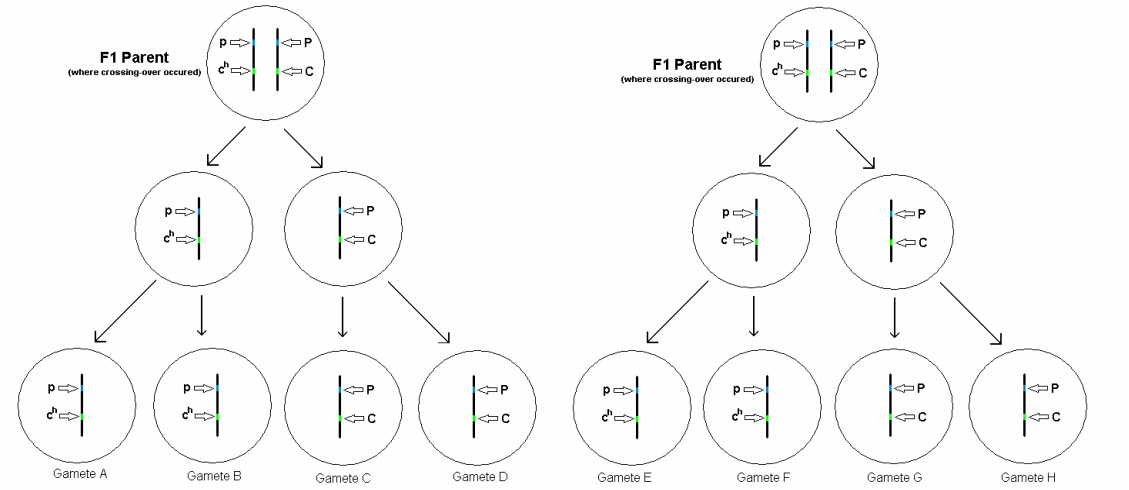
Let's assume this happens during meiosis in our amber × agouti point siamese cross (see figure 14). Say, crossing over occurs there gametes C and F combine (see figure 13).

Instead of getting a F1 agouti offspring with the genotype A//A C,p//c^h,P (as shown in figure 14), you now have a F1 offspring with the genotype A//A C,P//c^h,p (as shown in figure 13).

If by change you get two agouti F1 offspring of opposite sexes where crossing-over occurred in both offspring and you cross these two F1 agoutis with each other, you should get amber point siamese (see figure 15):

		F1 agouti parent (crossing-over)	
		A/ C,P/	A/ c^h,p/
F1 agouti parent (crossing-over)	A/ C,P/	A//A C,P//C,P Agouti	A//A C,P//c^h,p Agouti
	A/ c^h,p/	A//A C,P//c^h,p Agouti	A//A c^h,p//c^h,p Amber point siamese

Yet, to get an amber point siamese by crossing-over is still very unlikely. This is because firstly *both* chromosomes of chromosomal pair 1 have to break *simultaneously* and then the wrong parts have to grow together. And even after this, crossing-over have to occur in both a male and a female F1 baby and then to stack the odds even more against the possibility of an amber point siamese,



you will have to cross the right two F1 babies together and you won't be able to physically distinguish between F1 offspring where crossing-over occurred and where it did not (all F1 offspring still have an agouti phenotype, thus they all look the same).

Figure 15: *The process of meiosis with crossing over occurring for the F1 agouti × F1 agouti cross*

And to even make it more unlikely to breed an amber point siamese, the break in the chromosomes have to occur between the **P** and the **C** genes. If the **P** and the **C** genes are located very close to each other the chances are less likely that the break in the chromosome will occur between these two genes, than if the **P** and the **C** genes were located far from each other. Figure 16 shows what happens when the two genes are located close to each other and when the break in the chromosomes does not occur between those two genes, the resulting F1 agouti still essentially have the same genotype when considering only the **P** and the **C** genes.

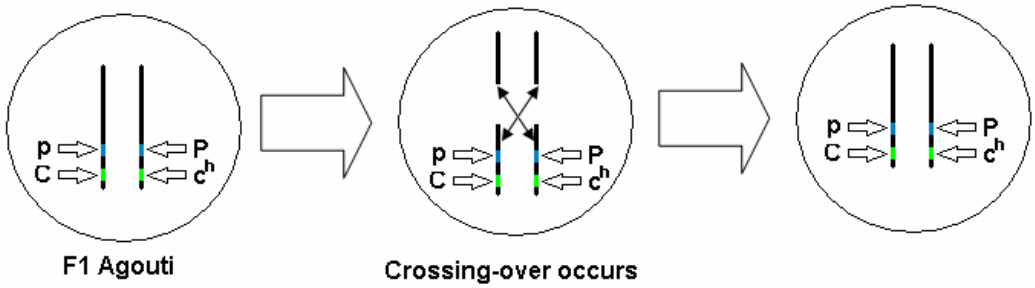


Figure 16: Crossing over occurs for a F1 agouti, but break does not occur between the two genes

4.8 Polymer factors

In reality no genetic characteristic is controlled by only one gene. A gene's expression is influenced to some extent by countless other genes with individual effects often so slight that they are very difficult to locate and analyse (p 302) (Keeton 1973).

These "other" genes work together in groups to form a certain phenotype, say you need four recessive genes to form a physically large rat. If you mate this large rat to a small rat you might get medium sided rats, but if you mate those offspring together again it is very unlikely that you will get a large rat again. If large size is controlled by four genes, the chances for a large rat in the F2 generation would be 1 in 256! But what if large size is not only controlled by four genes, but by 6 or 8 or even more, the chances for a large rat now becomes so small in the F2 generation that it is not worth the while to try and calculate it. The other problem is that one of these genes playing a role in forming a large rat has such a minor affect on the rat's phenotype that it is impossible to know what genotype you have by just looking at a certain rat.

These groups of genes are termed polygenes or polymer factors and apart from genes that control size in the rat, there are many other groups of genes controlling aspects such as face shape, body shape, size, etc. There are also polygenes that affect the hue or intensity of colour which work in addition to the basic colour variety.

These genes result in that, for instance not each and every champagne are exactly the same shade of colour as another individual although both rats are still champagnes. Other polygenes will work in addition to the basic marking genes such as hooded (**h**) and affect the markings by controlling where white areas should be and how much white areas there should be. That is why no one hooded rat looks exactly the same as another hooded, one hooded rat might have a perfect straight line down the back while another might have a uneven line.

Thus, when breeding rats, polymer factors are normally not considered in calculating the phenotypes of the offspring. Factors such as size are normally obtained rather through selective breeding that by planned genetic breeding as it is almost impossible to keep tract of the polymer factors involved.

4.9 Penetrance and expressivity

Back in section 4.2.2 we discussed dominant factors, but we must not assume that a dominant factor will always express the phenotypic effect of that gene. Some dominant factors depend on other genes in order to express phenotypically and when such a gene is expressed it can also show many different degrees of intensity. Therefore we refer to the penetrance and expressivity of that gene. Penetrance is the percentage of individuals that, when carrying a given gene in proper combination for its expression, actually express the gene's phenotype and expressivity denotes the manner in which the phenotype is expressed (Keeton 1973).

The autosomal dominant pearl gene (**Pe**) of the rat is such a gene, it can only be expressed on a mink background, not on a normal black background as one would expect. Thus a a//a M//M Pe//pe will be a black rat and not a pearl rat as expected, but a//a m//m Pe//pe will be a pearl rat. Therefore, the pearl gene (**Pe**) is dependant on the mink gene (**m**) in order to express phenotypically.

If you mate a pearl rat (a//a m//m Pe//pe) to a black rat carrying mink (a//a m//m pe//pe), you will get a ratio of 2:1:1 black; pearl; mink instead of 1:2:1. Thus the penetrance is 25% (or 1 out of 4) for pearl.

		Pearl	
		m/ Pe/	m/ pe/
Black	M/ pe/	M//m Pe//pe Black	M//m pe//pe Black
	m/ pe/	m//m Pe//pe Pearl	m//m pe//pe Mink

Pearl's also come in a wide range of shades, ranging from light coloured pearls to darker more brownish coloured pearls. Therefore we say the expressivity of pearl is variable.

Penetrance and expressivity can also be influenced by environmental factors (Keeton 1973). For instance, the siamese colour is influenced by temperature. A siamese raised in a cold environment will be darker than one raised in a warm environment. Also if a siamese looses a patch of fur on his back for instance (where the fur is normally a cream colour) it will grow back darker than normal as the piece of bare skin was exposed and therefore at a slightly lower temperature than the rest of the skin which was covered in fur.

A rat which has the genetic potential to be a large rat, won't be large if he is starved or not fed a properly balanced diet. Therefore, as we can see the expression of genes depend on other genes present and the environment. Genes only give the potential for a certain characteristic and these other factors can determine if those potential will be realized.

4.10 Lethal factors

We know that when a fault occurs on a gene, a mutation can result (two faulty genes are needed for a recessive mutation and only one for a dominant mutation). Yet the mutated individual is always deficient is some way or another when compared to the wildtype form, e.g. an amber rat is deficient in black-brown pigments while the agouti isn't. Sometimes a mutation can cause such a great deficiency that the individual cannot function and dies. Genes which cause such mutations are termed lethal.

To take a simple example, a plant needs chlorophyll (the green pigment in its leaves) to photosynthesize (make food for itself) and thereby survive. If a mutation occurs that causes the plant not to have any chlorophyll, the plant will die as it cannot photosynthesize.

In the rat fancy, the well known pearl gene (**Pe**) can result in a lethal phenotype. The pearl gene (**Pe**) is an autosomal dominant gene that causes pearl (a//a m//m Pe//pe) and cinnamon pearl (A//- m//m Pe//pe[8]) coloured rats when only one copy of the gene is present, but when there are two copies of the pearl gene the homozygous pearl (Pe//Pe) embryos get aborted by the mother rat as they are no longer viable and cannot grow into adult rats. The pearl gene is however termed semi-lethal as it is only lethal when homozygous (Pe//Pe) and not when heterozygous (Pe//pe).

4.11 Pleiotrophy

Sometimes a gene causes more than one phenotypic effect (pleiotrophy) (Keeton 1973). In the rat fancy overseas[9] there is an autosomal dominant gene, termed white spotting, **Ws** (Rat biology 2004c) that causes a certain kind of white spotting in the rat. This gene are often used to breed American type husky rats, blazed rats and odd-eyed rats depending with which other marking and colour genes it is combined with. Yet, this gene also causes megacolon, an extremely horrible and dangerous disease. Markings caused by this specific white spotting gene are often referred to as high risk whites[10] or just high whites.

Note that there are also many other types of white spotting genes that can cause markings such as blazed in the rat. Therefore not every blazed rat is at risk of developing megacolon, just the ones caused by this white spotting gene (**Ws**). Another myth of megacolon is that you can breed it our of a line, this is not true, as megacolon is directly linked to dominant white spotting and therefore cannot be bred out of dominant white spotting type rats (Brooks 2005). If you do not want megacolon in your lines do not then introduce the white spotting gene into your lines.

5. Conclusion

The knowledge of genetics provides a powerful tool for anyone breeding rats or any other animal. However, a lot of patience is also required to breed the more difficult varieties caused by multiple genes. It can take a few years for instance to breed a Russian silver point siamese as you will have to wait for each consecutive generation to reach maturity to breed from them and sometimes certain crosses will have to be redone when a certain required variety did not occur in a particular litter. Yet, even when one wants to breed various varieties, one still has to breed ethically. Never use a sick or a malformed individual for breeding no matter how beautiful its colour is, and never breed more rats than you can take responsibility for. To breed even one rat of a nice colour will also produce a large number of rats with more common colours which you might not find homes for.

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Notes:

1) The agouti and black parent rat in this case is known as the parental generation (P generation), their offspring as the first filial generation (F1 generation) and the offspring of the F1 generation as the F2 generation, etc. (Keeton 1973).

2) Normally genetic formulas are only written with one forward slash (/) between the genes, but for this article we will use two to indicate a genotype formula and one to indicate a gamete.

3) When we say a non-rex rat cannot carry the rex factor we are referring to the commonly found dominant rex mutation. There is another very rare *recessive* rex mutation, where a non-rex rat can actually carry this recessive rex factor (however, currently neither dominant nor recessive rex is currently found in South Africa).

4) It is sometimes stated that male tortoiseshell cats also occur, but this is not caused by normal inheritance but when a mistake occurs upon fertilization resulting in an abnormal cat where 3 gametes fused (two female and one male) instead of two. This gives a cat with 3 sex chromosomes (XXY) instead of two. If one of the X chromosomes in this case had the mutated O gene and the other non mutated o, this “male” cat will be tortoiseshell. Such male cats are often sterile. (Giant book of the cat 2002).

5) A locus refers to the position of the gene on the chromosome (Grant 2006).

6) Actually there is also a new fourth allele, the one giving our South African sable siamese. The sable siamese allele also seems to be co-dominant to $\mathbf{c}^{\mathbf{h}}$ and **c**.

7) Litter outcomes can be expressed in various ways; one can use ratios, percentages or fractions. All of these basically express the same thing.

8) A dash (-) is often used to indicated that a particular gene can be either the one or another other allele. A//- therefore indicate either a A//A or an A//a genotype.

9) This type of dominant white spotting does NOT occur in South Africa, therefore no South African rat are at risk of developing megacolon.

10) This term sometimes leads to confusion as it does not refer to completely white rats, but any variety caused by dominant white spotting, even it the variety has mostly coloured areas compared to white areas (e.g. blazed Berkshires caused by dominant white spotting).

I would like to thank J.C. Combrink for proofreading this article. If you have any questions about this article, varieties or anything relating to genetics, you are welcome to contact me at aurora@rattyrascals.co.za or by posting questions on the S.A. Rat Fan Club forum at http://www.rattyrascals.co.za/chatrat/