PERSPECTIVES

William Bateson: a biologist ahead of his time

PATRICK BATESON*

Sub-Department of Animal Behaviour, University of Cambridge, Madingley, Cambridge CB3 8AA, UK

William Bateson coined the term genetics and, more than anybody else, championed the principles of heredity discovered by Gregor Mendel. Nevertheless, his reputation is soured by the positions he took about the discontinuities in inheritance that might precede formation of a new species and by his reluctance to accept, in its full-blooded form, the view of chromosomes as the controllers of individual development. Growing evidence suggests that both of these positions have been vindicated. New species are now thought to arise as the result of genetic interactions, chromosomal rearrangements, or both, that render hybrids less viable or sterile. Chromosomes are the sites of genes but genes move between chromosomes much more readily than had been previously believed and chromosomes are not causal in individual development. Development, like speciation, requires an understanding of the interactions between genes and the interplay between the individual and its environment.

Introduction

William Bateson is credited with coining the term 'genetics' as it is currently used as a noun. The adjective 'genetic' had been introduced into the English language in the 1830s. When Charles Darwin was nominated for the Royal Society's highest award, the Copley Medal, his supporters referred, among other things, to his contribution to 'genetic biology'. Darwin was eventually awarded the Copley Medal in 1864, but mention of this contribution was omitted as grounds for making the award by a timorous Council of the Royal Society because *On the origin of species* was still so controversial (Burkhardt 2001). It is clear in the usage applied to Darwin's then disputed contribution that 'genetic' meant 'pertaining to origins'. Anyhow, genetics as a noun, meaning the study of heredity and variation, was first used by William Bateson in a letter to Adam Sedgwick in 1905 when he hoped to be appointed to a new chair (B. Bateson 1928).

William Bateson was the most vigorous promoter of Mendel's ideas at the beginning of the twentieth century and effectively launched the modern subject of genetics. Historians of biology acknowledge the importance of this contribution but criticise his ideas on sudden changes in evolution leading to the origin of new species and his questioning of the role of chromosomes (Mayr 1982). In this article I re-examine these criticisms of Bateson in the light of modern advances in biology.

Bateson was born on 8 August 1861. He was raised in a comfortable home and had an eminent father who was for 24 years Master of St John's College, Cambridge. Family life was filled with conversation and William and his siblings grew up to be strong-willed, disputatious and highly intellectual. William obtained a first class in natural sciences at Cambridge, specializing in zoology. His first major study was on the embryology of *Balanoglossus* living on tidal mudflats; it looks like a worm but is now regarded as a vertebrate ancestor. It was particularly common in Chesapeake Bay on the east coast of the United States and it was there that Bateson got to know a brilliant American zoologist, William K. Brooks, who was bringing out a book about heredity at the time (Brooks 1883). Although few biologists doubted that Charles Darwin had provided the most coherent and complete explanation for adaptation by the process of natural selection, the necessary conditions for one species to become distinct from another remained a source of dispute.

Discontinuous variation

Darwin's mechanism for evolutionary change consisted of three crucial steps. Each step must have been in place if adaptation by the organism to the environment occurred in the course of biological evolution. First, variation must have existed. Second, some variants must have survived more readily than others. Third, the variation must

*E-mail: ppgb@cam.ac.uk. Correspondence to Prof. Patrick Bateson, The Provost's Lodge, King's College, Cambridge CB2 1ST, UK.

have been inherited. While Darwin's proposal provided a powerful and plausible mechanism for generating adaptations, it was less obvious that it would provide what was needed for the formation of a new species. His friends Thomas Henry Huxley and Francis Galton were doubtful. In his excellent book, Donald Forsdyke (2001) describes how George Romanes developed a physiological theory for hybrid sterility (e.g. Romanes 1886). The theory was intended to solve the speciation problem, but it was roundly attacked by Darwin's defenders and Romanes's contributions to evolutionary theory were forgotten, although his book *Animal intelligence* is read to this day. William Brooks was also doubtful whether speciation could be explained by natural selection. Much of this scepticism rubbed off on Bateson in the course of their long discussions after work in Chesapeake Bay.

In the first phase of his life's work, Bateson wanted to know what variation within a species might look like. He was particularly interested in finding major discontinuities in characters. He amassed a great quantity of examples in his book *Materials for the study of variation* which appeared in 1894 (W. Bateson 1894). He believed that such discontinuities could provide evidence for steps that might lead to the appearance of a new species. This interest prepared him for the rediscovery of Mendel's work which provided the rules for how the qualitative differences between members of the same species could be inherited. Bateson was sometimes thought to be anti-Darwinian because of his search for discontinuities in natural variation (Bowler 1983). Mark Ridley (1996) has argued that Darwin himself was only a gradualist as far as adaptation was concerned and that he had not nailed his colours to the mast over speciation. Anyhow, Bateson was never opposed to the Darwinian proposal for the evolution of adaptations, although he did not share the belief, usually attributed to Darwin, that the evolution of a new species always involved continuous and gradual modification.

Principles of inheritance

Having established that discontinuities were to be found in nature, the next step was to discover what happened to

Figure 1. William Bateson (1861–1926).

such discontinuities from one generation to the next. Bateson set to work on the experimental breeding of animals and plants to find out how variations might be inherited. Before the rediscovery of Mendel's work, the best known principle was the Law of Ancestral Heredity, promulgated in the nineteenth century most actively by Francis Galton (1897). Those of a mathematical bent liked it because it meant that a prediction about the characteristics of an individual could be derived probabilistically from the characteristics of the ancestors. Galton produced two forms of the Law of Ancestral Heredity, but these were mathematically inconsistent (Provine 1971). Karl Pearson subsequently cleaned up the mathematics (Pearson 1898) but did not help matters when he called his revision 'Galton's theory'. Confusion reigned. The independent rediscovery of Mendel's work by De Vries, Tschermak and Correns changed all that. An enjoyable account is given by Henig (2000). When Bateson became aware of Mendel's work he realized, supposedly in a flash, that here were the principles that would make sense of heredity. Supposedly, he was reading a paper by De Vries on a train when he was travelling from Cambridge to London to address the Royal Horticultural Society. It is worth noting that Olby (1987) has expressed some scepticism about both the suddenness and the site of this realization.

Mendel's discoveries may be summarized as follows. Inherited factors influencing the characteristics of an organism come in pairs; while this is usually true, sex linkage (arising from unequal pairings of the sex chromosome in male mammals and female birds) was only discovered later. One factor is often dominant to the other and the recessive one lies latent within an individual. It is a matter of chance which one enters into the gamete that fuses with the gamete of the other parent. The particular factor that enters a gamete is usually unrelated to the member of another pair of factors; the linkage of some factors was only to emerge later.

Some have suggested that, if Mendel's work had not been rediscovered, Bateson would have derived the same principles of inheritance and, as J. B. S. Haldane put it, Bateson's body would now rest in Westminster Abbey. Alan Cock (1973) argued that this was unlikely, believing that by 1900 Bateson had, in his experimental work, '. . . reached an impasse which was resolved when he came to learn of Mendel'. And once that had happened, Cock goes on, Bateson was peculiarly well-fitted to recognize the importance and far-reaching implications of Mendel's work—as was seen, for example, in his appreciation of the work of a distinguished physician and contemporary, Archibald Garrod.

Garrod had become particularly interested in a rare abnormality in which a person produces urine that blackens on exposure to air. It is highly noticeable at an early stage in life because the affected babies' nappies are stained deeply by the black urine. The critical compound in the urine of people with alkaptonuria, as the condition is called, is homogentisic acid, of which 2.5 to 6.0 grams is produced each day. Alkaptonuria is much more common in men than in women and, though always rare, is particularly likely to occur in the offspring of first cousin marriages. Bateson got to hear of this and in December 1901 he and Saunders reported Garrod's finding to the Evolution Committee of the Royal Society. They wrote: '. . . the mating of first cousins gives exactly the conditions most likely to enable a rare and usually recessive character to show itself. If the bearer of such a gamete mates with individuals not bearing it, the character would hardly ever be seen; but first cousins will frequently be bearers of *similar* gametes, which may in such unions meet each other, and thus lead to the manifestation of the peculiar recessive characters in the zygote'. (Royal Society. Reports to the Evolution Committee 1902)

Alexander Bearn, biographer of Garrod (Bearn 1993), found a long letter from Garrod to Bateson dated 11 January 1902 beginning: 'It was a great pleasure to receive your letter and to learn that you are interested in the family occurrence of alkaptonuria.' This suggests that Bateson initiated the correspondence. Anyway, Garrod was quick to see the significance of Mendelism for congenital human conditions and referred explicitly to this insight in his next paper (Garrod 1902). No case of two alkaptonurics having children together was known. Garrod had both male and female alkaptonuric patients and he used to get them into the ward at the same time in the hope that they might become fond of each other, marry and have children (Bearn 1993). If the children had been alkaptonuric, that would have clinched the Mendelian hypothesis. That was not to happen but Garrod, with his concept of the inborn errors of metabolism, is now regarded as the founder of biochemical genetics.

Mendelians and biometricians

In 1902 Bateson renewed an intellectual battle with the biometrician Walter Frank Raphael Weldon. This bitter struggle had started when his former friend and mentor wrote a critical review of *Materials for the study of variation* eight years earlier. Weldon now tried to argue that Mendel simply described a special case and, in any event, the results could be explained in terms of the Law of Ancestral Heredity derived from Galton. This patronizing comment fired up Bateson who, with his enormous energy and determination, wrote in a few months a fierce rebuttal of Weldon's review in the book called *Mendel's principles of heredity: a defence* (W. Bateson 1902). Weldon had further infuriated Bateson by concluding his review of Mendel's paper by writing that, without wishing to belittle Mendel's achievement, he wanted 'to call attention to a series of facts which seem to me to suggest

fruitful lines of enquiry'. Bateson commented that Weldon was about as likely to kindle interest in Mendel's discoveries as light a fire with a wet dishcloth.

When Weldon suddenly died in 1906, Bateson wrote to his own wife Beatrice of this fierce squabble with his former friend, 'If any man ever set himself to destroy another man's work, that did he do to me . . . ', but in another letter wrote: 'To Weldon I owe the chief awakening of my life. It was through him that I first learnt that there was work in the world which I could do. . . . Such a debt is perhaps the greatest that one man can feel towards another; . . . . ' (B. Bateson 1928). Shaken by Weldon's death, Bateson offered an olive branch to Karl Pearson, the principal biometrician of the time, but the peace offering was rejected and the battle between the Mendelians and the biometricians persisted.

Bateson's common-sense rejection of the biometricians' premature attempts to formalize the principles of heredity does seem justified now. R. A. Fisher believed that '. . . had any thinker in the middle of the nineteenth century undertaken, as a piece of abstract and theoretical analysis, the task of constructing a particulate theory of inheritance, he would have been led, on the basis of a few very simple assumptions, to produce a system identical with the modern scheme of Mendelian or factorial inheritance' (Fisher 1930, p. 7). This assertion overplayed the power of mathematical modelling. Nobody, apart from Mendel, had been led to the deduction that inherited factors influencing the characteristics of an organism come in pairs—or at least that they usually do. No amount of clever mathematics could have led to the deduction that one is often dominant to the other. (Galton understood that many 'gemmules', as Darwin had called them, capable of influencing the characteristics of an organism, must often lie latent, but that wasn't a mathematical deduction—it was based on empirical observation.) No amount of clever mathematics could have led to the deduction that it is a matter of chance which of each paired factors enters into the gamete that fuses with the gamete of the other parent. And no amount of clever mathematics could have led to the deduction that the particular factor that enters a gamete is usually unrelated to the member of another pair of factors. Once known, the stage would be set for formalization—but not before.

For all that, it was a mathematician, Udny Yule, who pointed out at an early stage (Yule 1902) that the struggle between Bateson and the biometricians was entirely unnecessary since Mendelian factors could give rise to small changes and therefore be compatible with the view that evolutionary change was continuous. The differences between the Mendelians and the biometricians was primarily over whether discontinuous change could occur. Evidence of discontinuity, provided by 'sports'—strikingly different phenotypes—was regarded by the Mendelians as evidence against Darwin. When Fisher finally demonstrated to universal satisfaction that Mendelism could be reconciled with Darwin's notion of continuous evolutionary change, the source of the controversy seemed to have been removed (Fisher 1930). Fisher remained, however, fiercely critical of Bateson (Cock 1973). It is worth noting that, even though Fisher's resolution of the biometrician–Mendelian controversy has become the 'official' history, already in 1902 Bateson and Saunders had written as follows (Royal Society. Reports to the Evolution Committee 1902, pp. 152–153):

> It must be recognized that, in for example, the stature of a civilised race of man, a typically continuous character, there must certainly be on any hypothesis more than one pair of possible allelomorphs. There may be many such pairs, but we have no certainty that the number of such pairs, and consequently of the different kinds of gametes, are altogether *unlimited* even in regard to stature. If there were even so few as, say, four or five pairs of possible allelomorphs, the various homo- and hetero-zygous combinations might, on seriation, give so near an approach to a continuous curve, that the purity of the elements would be unsuspected, and their detection practically impossible.

Fisher, Sewall Wright and Haldane all brought mathematical rigour to the subject and founded the field of theoretical population genetics. While they agreed upon the importance of Darwinian evolution, each of them produced a distinct model and some inconsistencies between the theoretical frameworks on which their subject is based remain to this day (Provine 1971). From the standpoint of Darwinian theory, Ernst Mayr (1942) persuasively argued that small isolated populations could rapidly evolve distinct characteristics that made them genetically incompatible with closely related populations and thus form a new species. From this perspective, it might seem in hindsight that Bateson's search for discontinuities in order to explain the origin of species was a waste of time. However, the debate is far from over and, indeed, a view is growing that Bateson has been unjustly maligned (Forsdyke 2001).

Sudden changes in evolution

Discontinuity in evolution has been given special prominence by some modern palaeontologists who have been impressed by periods of stasis and sudden change in the fossil record (Eldredge 1995; Gould 2002). They suggest that after periods of stasis in evolution sudden changes can occur in the fossil record and these may represent the appearance of new species. This idea of discontinuity has recurred periodically and, notably, was foreshadowed in the writings of Goldschmidt (1940), who, in a memorable phrase, referred to a fresh arrival that might give rise to a new species as a 'Hopeful Monster'.

Long before Goldschmidt, Galton (1892, pp. 354–355) had produced a vivid image of how the law of continuity

in evolution might be satisfied by a series of changes in jerks.

> The mechanical conception would be that of a rough stone, having, in consequence of its roughness, a vast number of natural facets, on any one of which it might rest in 'stable' equilibrium. That is to say, when pushed it would somewhat yield, when pushed much harder it would again yield, but in a less degree; in either case, on the pressure being withdrawn it would fall back into its first position. But, if by a powerful effort the stone is compelled to overpass the limits of the facet on which it has hitherto found rest, it will tumble over into a new position of stability, whence just the same proceedings must be gone through as before, before it can be dislodged and rolled another step onwards.

Hosts of examples of big events having no effect and small events leading to big changes are to be found and many of these are now formalized by the nonlinear mathematical techniques derived from catastrophe theory and chaos.

Even though the discontinuities in natural variation, which Bateson had documented so carefully, no longer pose a problem in developmental biology, Hopeful Monsters are disparaged to this day on the grounds that, even if a big change in the phenotype could occur as a result of mutation, the Hopeful Monster would be a novelty on its own with no possibility of finding a mate. Without a mate it could never found a new species. However, if, somehow or other, enough Hopeful Monsters sprang into existence simultaneously and thus were able to breed successfully with each other, the possibility exists of competition between the Hopeful Monsters and the stock from which they sprang. It is not at all difficult to suppose that, by the process of natural selection, Hopeful Monsters could quickly replace their competitors if they were better adapted to the environment. No new fancy principles of evolution are involved here.

The critical issue for evolution is: how could a whole group of individuals, founders of a new species, suddenly arise at the same time? I have a modest proposal which I first buried in an obscure publication nearly 20 years ago (P. Bateson 1984). Suppose that a population splits into two subgroups as the result, say, of migration. The subgroups remain separated for many generations and a different mutation goes to fixation in each of them. Then, the populations merge again. The combination of the mutated genes in the two previously separated subgroups interacts to produce a radically new phenotype which is sufficiently frequent in the population to allow breeding to occur. Now the conditions are in place for a competition between the phenotypes. If the new phenotype is more successful than the old, the historical record would show a discontinuity in evolution. The argument is presented visually in figure 2.

At the time I first suggested this idea, I was unaware of the Dobzhansky–Muller model for speciation, which has points of similarity and suggests that postzygotic isolation results from an interaction between two or more genes (Orr and Presgraves 2000). Suppose the initial genotype is *aabb*, the population splits and in one population an *A* mutation appears and goes to fixation and in the other population a *B* mutation appears and also goes to fixation. If *A* and *B* do not function well together, then hybrids between the two populations will be less viable or infertile. As Orr and Presgraves (2000) point out, this model highlights the role of epistasis in evolution. Though credit is usually given to Dobzhansky (1937) and Muller (1940), it is highly relevant to my argument that, as Orr (1997) notes, the problem was first solved by Bateson. This is what he wrote (W. Bateson 1909, pp. 97–98):

> When two species, both perfectly fertile severally, produce on crossing a sterile progeny, there is a presumption

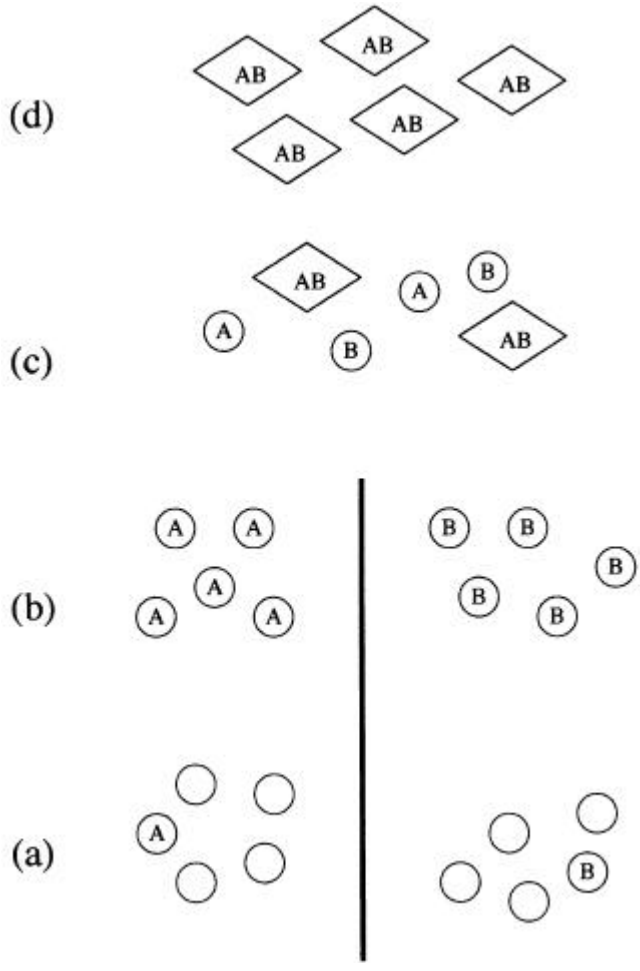

Figure 2. A proposal for how 'Hopeful Monsters' might evolve (from Bateson 1984). Time runs upwards from the bottom. (a) Appearance of two different mutations (*A* and *B*) in separated populations of the same species. (b) The mutations spread and go to fixation. (c) The populations merge and the interaction between *A* and *B* gives rise to a new phenotype. (d) The new phenotype (with the *AB* genotype) is more successful than the old one and replaces it.

> that the sterility is due to the development in the hybrid of some substance which can only be formed by the meeting of two complementary factors. That some such account is correct in essence may be inferred from the well-known observation that if the hybrid is not totally sterile but only partially so, and this is able to form some good germ cells which develop into new individuals, the sterility of these daughter individuals is sensibly reduced or may be entirely absent. The fertility once re-established, the sterility does not return in the later progeny, a fact strongly suggestive of segregation. Now if the sterility of the cross-bred be really the consequence of the meeting of two complementary factors, we see that the phenomenon could only be produced among the divergent offspring of one species by the acquisition of at least *two* new factors; for if the acquisition of a single factor caused sterility the line would then end. Moreover each factor must be separately acquired by distinct individuals, for if both were present together, the possessors would by hypothesis be sterile. And in order to imitate the case of species each of these factors must be acquired by distinct breeds.

It seems clear that priority for the speciation model must be given to Bateson. The irony is all the greater because Bateson is often portrayed by his critics as having no understanding of evolution.

My conjecture about a means for the sudden emergence of discontinuous change in evolution does not necessarily imply speciation. However, it would do so if hybrids between old and the new phenotypes were less viable. A plausible case would be a change in chromosome number which could prohibit the formation of gametes in hybrids. Examples of closely related species with different numbers of chromosomes are well known. In horses the chromosome number ranges from 32 in *Equus zebra hartmannae* and 46 in *Equus grevyi,* to 62 in *Equus assinus* and 66 in *Equus przewalski*; all but two of the horse hybrids are sterile (King 1993). Max King concludes his book (King 1993, p. 288): 'Chromosomal studies of both plants and animals indicate that many species have evolved by chromosomal speciation . . . .'

The chromosome theory

Bateson had been castigated for his slowness in understanding the chromosome theory, which so neatly explained segregation and linkage, and for even partially retracting his conversion in the last year of his life (Crowther 1952). For this he was 'relegated to the back lots of scientific history' (Henig 2000). Another modern commentator (Tudge 2000) wrote: 'William Bateson, though a pioneer of the new genetics, seemed positively distraught by the thought that the chromosomes might actually carry the genes (for reasons I confess I cannot fathom).'

Why did Bateson so oppose the chromosome theory? Here was a further mechanism that matched perfectly to the principles established by Mendel which Bateson had fought so hard to defend. Was this simply conservatism as Coleman (1970) suggests? I can only guess at an alternative view, but I have a strong hunch that Bateson was irritated about the glibness of the explanation offered for the role of chromosomes. The chromosome was portrayed not simply as a structural site of the gene but almost as the gene itself. Worse, the chromosome was treated as the pre-formed version of the character influenced by the gene on that chromosome.

The cytology of chromosomes had been described since the 1880s (Mayr 1982). Bateson must have known chromosome numbers and structures varied, even between closely related species. How could the structure that was supposed to control development be changed by itself? Something has to be doing it. Nowadays we are not worried by the thought that chromosome number and structure can be influenced by genes because we have separated in our minds a particular strand of DNA molecule from the structure on which it sits at the time of cell division. We are also aware of how DNA can move around the chromosomes in a remarkable way. Nearly half of the human genome consists of transposable elements (Dover 2000). Finally, a clear distinction is now made between structure and development. Sydney Brenner (2001) noted that no less a man than Erwin Schrödinger (1994) could write: 'The chromosome structures are at the same time instrumental in bringing about the development they foreshadow. They are code law and executive power, or to use another simile, they are the architect's plan and the builder's craft in one.' Brenner refers to this image as 'Schrödinger's fundamental error' and goes on (Brenner 2001, p. 34): 'The chromosomes contain the information to specify the future organism and a description of the means to implement this, but not the means themselves.'

Whether or not Bateson had properly articulated Brenner's distinction in his own mind is debatable, but his intuition would have made him suspicious about the overselling of the role of chromosomes. Also, Bateson's sense that the degree of organization required for the development of an adult organism would not be represented by single particles has a remarkably modern, post-genomic feel to it. The criticism of him for failing to foresee the future of twentieth century molecular biology results from a confusion that has run through these discussions for the past 100 years. The structures, required for the transmission from one generation to the next through the gametes, should be distinguished from the developmental systems that lead to the expression of the characters of the whole organism.

Relatively early in his professional career Bateson had an inchoate notion of what he called a 'vibratory theory' of development. In an excited letter to his sister Anna, dated 14 September 1891, he wrote: 'Divisions between

segments, petals etc are *internodal* lines like those in sand figures made by sound, i.e. lines of maximum vibratory strain, while the mid-segmental lines and the petals, etc. are the *nodal* lines, or places of minimum movement. Hence all the *patterns* and *recurrence of patterns* in animals and plants—hence the perfection of symmetry—hence bilaterally symmetrical variation, and the *completeness* of repetition, whether of a part repeated in a radial or linear series etc., etc.' (B. Bateson 1928, p. 43). This idea stayed with him and he wrote about it more extensively in *Problems of genetics* (W. Bateson 1913) and was clearly thinking about these issues into the last year of his life. It is not necessary to accept his developmental theory to understand that he could have been mistrustful of claims that in effect collapse the gene with the chromosome, on the one hand, and with the phenotypic character it influences on the other. It is interesting that Mayr (1982), in his critique of Bateson, is inconsistent. He writes (p. 736) that Bateson '. . . failed to make a clear distinction between an underlying genetic factor and the resulting phenotypic character'. Later (p. 772) he describes Bateson as a 'physicalist' who favoured dynamic explanations based on physics and was horrified by what seemed to be the revival in chromosome theory of '. . . preformation in a modernised form'. Quite possibly Bateson was ambivalent but, in my view, his style would be now called an unformalized systems approach and he had moved a long way from the one gene : one phenotypic character thinking that still dogs some modern writing. The relevance of Bateson's style of thinking to modern biology seems ever more obvious (Bock and Goode 1998; P. Bateson and Martin 1999).

Bateson was obviously aware of the interaction between genes. He did a famous experiment with Punnett in which he crossed two white strains of chicken, White Silkies with White Dorkings. The offspring were not white. They were coloured, so it became clear that something came from a White Silky parent that interacted with something else that came from the White Dorking and it was this interaction that produced the colour. Even the archetypal case for inborn errors of metabolism suggested an interaction. Alkaptonuria was reported as being much more common in men than in women (Garrod 1902). If this were not simply that men were more likely to bring their condition to the attention of a medical doctor, the sex difference would indicate that the expression of the recessive gene was affected by another gene on the Y chromosome.

Once epistasis is recognized as important in the developmental process, the factors influencing phenotypic characters are less profitably thought about in terms of the genes as units but in terms of the factors that are generated downstream. As Forsdyke (2001) notes, this was an issue about which Bateson was especially alert and he was already aware of how some factors might be the product of many things some of which are not genes. Even in the simplest case the interactions are not strictly between genes but between the products of genes.

Of the three great figures who started the formalization of population genetics, Sewall Wright was the most sensitive to epistasis (Wright 1930). Wright believed that selection for single genes was far less effective than the selection of interaction systems. In this way he was much more like Bateson than either of the other two great architects of theoretical population genetics. Fisher was keen to isolate the nonadditive effects in his equations so that he could deal with the much more tractable additive effects. However, the mathematical brilliance has arguably got in the way of understanding the biological phenomena. The point can be made by looking at the details of a modern example provided by people with the Kallmann syndrome.

The main behavioural consequence of the Kallmann syndrome in men is a lack of sexual interest in members of either sex. The syndrome is caused by damage at a specific genetic locus (Pfaff 1997). The syndrome was classically described as sex-linked but other genes that have been found to produce the same syndrome are autosomal. Cells that are specialized to produce a chemical messenger called gonadotropin-releasing hormone (GnRH) are formed initially in the nose region of the foetus. Normally the hormone-producing cells would migrate into the brain. As a result of the genetic defect, however, their surface properties are changed and the cells remain dammed up in the nose. The activated GnRH cells, not being in the right place, do not deliver their hormone to the pituitary gland at the base of the brain. Without this hormonal stimulation, the pituitary gland does not produce the normal levels of two other chemical messengers, luteinizing hormone and follicle stimulating hormone. Without these hormones, the testes do not produce normal levels of the male hormone testosterone. Without normal levels of testosterone, the man shows little sign of normal adult male sexual behaviour. Even in this relatively straightforward example, the pathway from gene to behaviour is long, complicated and indirect. Each step along the causal pathway requires the products of many genes and has ramifying effects, some of which may be apparent and some not.

In his last published paper, Bateson (1926) wrote that while the conception of linkage provided by the chromosome theory probably contains an essential truth, it is in some important respect imperfect. He goes on: 'What we know of the transmission of family likenesses both in physical and mental attributes is not easily consistent with the theory of random assortment in chromosome groups.' A child's characteristics are not a simple blend of its parents' characteristics. Most parents will find some particular likeness between themselves and their child. A daughter might have her mother's hair and her

father's shyness, for instance. The child may also have characteristics found in neither parent: a son might have the jaw of his grandmother and the moodiness of his cousin. The shuffling of discrete and supposedly inherited characteristics from one generation to the next is a commonplace of conversation. But what about a whole series of characters that sometimes create family likenesses? Are we simply captivated by a single shared characteristic or is something more interesting going on?

One idea that might explain family likenesses is that development is a dynamic and selective process. Genetic dominance depends a great deal on circumstances and a great many cells die in development. So it is possible that, throughout the process of formation of the body, the brain and the behaviour produced by the brain, those characteristics that develop are the ones that work best as an integrated whole. If this were the case, even sharing a very few genes that had a powerful controlling effect on development could lead to startling similarities. Whatever the explanation, Bateson's insight that an understanding of somatic development was needed to understand variation between individuals was way ahead of its time and demands the admiration of modern biologists.

Conclusion

Biology has moved a long way from the days when it was thought a good tactic to 'decouple' development of individuals from studies of evolution (Wilson 1976). The growth of the 'evo-devo' movement and developmental systems theory has pointed very strongly towards the benefits of bringing together these two branches of biology (Oyama *et al*. 2001). The study of speciation may be the area that is especially likely to be strengthened by this marriage (Arthur 2000). It is significant that Bateson started his professional life as an embryologist and clearly sensed the importance of this aspect of his early training. His belief that understanding the interactions involved in development are crucial for evolutionary theory is about to have its day.

The battle over discontinuous variation held up moves towards mathematical formalizations of genetics for many years. In his superb history of population genetics, William Provine (1971) felt that if, after Weldon's death, Bateson and Pearson had collaborated instead of fought, population genetics would have gained a significantly earlier start. That said, science is created by real people and argumentative, uncompromising Bateson was real enough for anybody. If he had been bland, Mendel would probably have remained unchampioned and might well have been disregarded. As the dust of these battles of the twentieth century settles, we start to see the advantages of bringing different approaches together and William Bateson re-emerges as a figure of great importance in the history of biology.

Personal note

I am not directly descended from William Bateson. He was the cousin of my grandfather and this relationship probably explains why, from a very early age, I told anybody who asked that I wanted to be a 'biologist' without having any clear idea what that might entail. Eventually I became a behavioural biologist (P. Bateson and Martin 1999), rather than a geneticist, but have, throughout my career, had a strong interest in both development and evolution. So in that sense I have retained a close intellectual link with William Bateson's heritage. The two sides of the family had a close social link because, when I was a boy, my parents cared for William's younger brother, Ned, when he was a widower and an old man. Everybody remarked on the astonishing resemblance between Ned and myself and, as I subsequently discovered years later, I also looked very much like William's son Gregory, even though he was only my second cousin once removed (see figure 3).

On the face of it, this article might seem like one Bateson leaping to the defence of another. That was not how I got started. When I first re-examined the work of my kinsman, I was preparing to deliver the Darwin Lecture of the Galton Society at the Royal Society of Medicine on 30 October 2001. I had been asked to compare William Bateson with Archibald Garrod as part of the hundredth anniversary of the rediscovery of Gregor Mendel's papers. Earlier in my career I had formed an unfavourable picture of William from talking to his younger brother, Ned, who called him a tyrant and one of the real Victorian autocrats. In a similar vein, his anthropologist son, Gregory, likened his father to the unbending Reverend Theobald Pontifex in Samuel Butler's *The way of all flesh*. Gregory, of course, was ambivalent and also spoke with fondness and respect about his father. Arthur Koestler, when writing *The case of the midwife toad*, though he had never met him, obviously took a strong dislike to William Bateson, whom he blamed for the suicide of the Austrian biologist Kammerer. Koestler's hero, Kammerer imagined that he had evidence for the inheritance of acquired characters until Bateson's devastating attack.

This disagreeable picture of William is strongly reinforced by many historians of science (Mayr 1982). As I delved into William Bateson's writings, the picture I had formed of him changed dramatically and I began to perceive somebody very different from the rude and stubborn controversialist. He was clearly beloved by those who worked closely with him and, as Forsdyke (2001) notes, his close colleague, Reginald Punnett, regarded him as dominant without ever being domineering (Punnett 1950): 'No one more delightful to work with than Bateson could be imagined.' In writing his biography of Gregory Bateson, David Lipset (1980) presented a

Figure 3. Family likeness in second cousins once removed. Gregory Bateson (1904–80) on the left was in his mid-40s at the time of the photograph. Patrick Bateson (1938–) on the right, a generation younger, was in his mid-30s when this photograph was taken. The probability of their sharing rare genes by common descent is 0.0156.

marvellously rounded picture of William. Certainly strong-willed and disputative, but also equipped with a strong sense of fun. Passionate about his science, he was also passionate about art. Coleman's (1970) description of him as conservative seemed odd to me until I remembered that Coleman was writing at a time when it was fashionable to explain scientific beliefs in terms of political ideology. Above all, as I read his work more closely, Bateson's ideas seemed to me to have a remarkably modern ring to them. I felt increasingly that William had been treated unfairly by both molecular and evolutionary biologists and that much of his thinking, inchoate though it often was, had been a long way ahead of his time.

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References

Arthur W. 2000 The concept of developmental reprogramming and the quest for an inclusive theory of evolutionary mechanisms. *Evol. Dev*. **2**, 49–57.

Bateson B. 1928 *William Bateson: naturalist*. Cambridge University Press, Cambridge.

Bateson P. 1984 Sudden changes in ontogeny and phylogeny. In *Behavior; evolution and integrative levels* (ed. G. Greenberg and E. Tobach), pp. 155–166. Erlbaum, Hillsdale, USA.

Bateson P. and Martin P. 1999 *Design for a life: how behaviour develops*. Cape, London.

Bateson W. 1894 *Materials for the study of variation: treated with especial regard to discontinuity in the origin of species*. Macmillan, London.

Bateson W. 1902 *Mendel's principles of heredity: a defence*. Cambridge University Press, Cambridge.

Bateson W. 1909 Heredity and variation in modern lights. In *Darwin and modern science* (ed. A. C. Seward), pp. 85–101. Cambridge University Press, Cambridge.

Bateson W. 1913 *Problems of genetics*. Yale University Press, New Haven.

Bateson W. 1926 Segregation. *J. Genet*. **16**, 201–235.

Bearn A. G. 1993 *Archibald Garrod and the individuality of man*. Clarendon Press, Oxford.

Bock G. R. and Goode J. A. E. (ed.) 1998 *The limits of reductionism in biology* (Novartis Foundation Symposium 213). Wiley, Chichester.

Bowler P. J. 1983 *The eclipse of Darwinism*. Johns Hopkins University Press, Baltimore.

Brenner S. 2001 *My life in science*. BioMed Central, London.

Brooks W. K. 1883 *The laws of heredity: a study of the cause of variation and the origin of living organisms*. Murphy, Baltimore.

Burkhardt F. 2001 Darwin and the Copley Medal. In *The correspondence of Charles Darwin*. Vol 12, 1864 (ed. F. Burkhardt,

D. M. Porter, S. A. Dean, P. S. White, S. Wilmot, S. Evans and A. M. Pearn), pp. 509–527. Cambridge University Press, Cambridge.

Cock A. G. 1973 William Bateson, Mendelism and biometry. *J. Hist. Biol*. **6**, 1–36.

Coleman W. 1970 Bateson and chromosomes: conservative thought in science. *Centaurus* **15**, 228–314.

Crowther J. G. 1952 *British scientists of the twentieth century*. Routledge & Kegan Paul, London.

Dobzhansky T. 1937 *Genetics and origin of species*. Columbia University Press, New York.

Dover D. 2000 *Dear Mr Darwin*. Weidenfeld & Nicolson, London.

Eldredge N. 1995 *Reinventing Darwin*. Weidenfeld & Nicolson, London.

Fisher R. A. 1930 *The genetical theory of natural selection*. Clarendon Press, Oxford.

Forsdyke D. R. 2001 *The origin of species revisited*. McGill-Queen's University Press, Kingston and Montreal.

Galton F. 1892 *Hereditary genius: an inquiry into its laws and consequences*, 2nd edition. Watts, London.

Galton F. 1897 The average contribution of each of several ancestors to the total heritage of the offspring. *Proc. R. Soc. London* **61**, 401–413.

Garrod A. E. 1902 The incidence of alkaptonuria: a study in chemical individuality. *The Lancet* 13 December, 1616–1620.

Goldschmidt R. 1940 *The material basis of evolution*. Yale University Press, New Haven.

Gould S. J. 2002 *The structure of evolutionary theory*. Harvard University Press, Cambridge.

Henig R. M. 2000 *A monk and two peas: the story of Gregor Mendel and the discovery of genetics*. Weidenfeld & Nicolson, London.

King M. 1993 *Species evolution: the role of chromosome change*. Cambridge University Press, Cambridge.

Lipset D. 1980 *Gregory Bateson: the legacy of a scientist*. Prentice Hall, Englewood Cliffs.

Mayr E. 1942 *Systematics and the origin of species*. Columbia University Press, New York.

Mayr E. 1982 *The growth of biological thought: diversity, evolution and inheritance*. Harvard University Press, Cambridge.

Muller H. J. 1940 Bearing of the Drosophila work on systematics. In *The new systematics* (ed. J. S. Huxley), pp. 125–268. Oxford University Press, Oxford.

Olby R. 1987 William Bateson's introduction of Mendelism to England: a reassessment. *Br. J. Hist. Sci*. **20**, 399–420.

Orr H. A. 1997 Haldane's Rule. *Annu. Rev. Ecol. Syst.* **28**, 195–218.

Orr H. A. and Presgraves D. C. 2000 Speciation by postzygotic isolation: forces, genes and molecules. *BioEssays* **22**, 1085–1094.

Oyama S., Griffiths P. E. and Gray R. D. (ed.) 2001 *Cycles of contingency: developmental systems and evolution*. MIT Press, Cambridge.

Pearson K. 1898 Mathematical contributions to the theory of evolution. On the Law of Ancestral Heredity. *Proc. R. Soc. London* **62**, 386–412.

Pfaff D. W. 1997 Hormones, genes, and behavior. *Proc. Natl. Acad. Sci. USA* **94**, 14213–14216.

Provine W. B. 1971 *The origins of theoretical population genetics*. University of Chicago Press, Chicago.

Punnett R. C. 1950 Early days of genetics. *Heredity* **4**, 1–10.

Ridley M. 1996 *Evolution*, 2nd edition. Blackwell Science, Oxford.

Romanes G. J. 1886 Physiological selection: an additional suggestion on the origin of species. *J. Linn. Soc. (Zool.)* **19**, 337–411.

Royal Society. Reports to the Evolution Committee 1902 *Report 1. Experiments undertaken by W. Bateson, FRS, and Miss E. R. Saunders*. Harrison & Sons, London.

Schrödinger E. 1944 *What is life? The physical aspect of the living cell*. Cambridge University Press, Cambridge.

Tudge C. 2000 *In Mendel's footnotes*. Cape, London.

Wilson E. O. 1976 Author's reply to multiple review of 'Sociobiology'. *Anim. Behav*. **24**, 716–718.

Wright S. 1930 Evolution in Mendelian populations. *Genetics* **16**, 97–159.

Yule G. U. 1902 Mendel's laws and their probable relations to intra-racial heredity. *New Phytol.* **1**, 193–207, 222–238.