surface between the *AB* corner and the *GH* corner and are approximately related by the pseudo-dyad axis of symmetry named dyad 1 by Cullis *et al.*[4]. However, the xenon atom in the α-chain lies nearer the *GH* corner and that in the β-chain closer to the *AB* corner. At first it might appear surprising that the xenon positions in the two chains are different from one another, and not the same as in myoglobin. The *AB* corners of the α- and β-chains differ in both the sequence and the number of residues they contain, giving rise to structurally different environments. The amino-acid sequences in the *GH* corners are also different, though here the two chains are of equal length. It should also be noted that the amino-acid sequences of myoglobin[6] and haemoglobin are quite different. Any change in atomic distribution near a cavity could easily change the electronic interaction with xenon towards an energetically unfavourable state.

The exact analysis of the xenon sites will have to await determination of the haemoglobin structure at high resolution. On the basis of Perutz's tentative atomic model of haemoglobin[7], the nearest neighbours of both xenon atoms are valine, leucine and phenylalanine. This complex is presumably stabilized, as in myoglobin, by dipole- and quadrupole-induced dipole and quadrupole moments and London interactions. A theoretical investigation by Kittel and Shore[8] of xenon polarizability has shown that the quadrupolar (as well as the dipolar) polarizability is particularly high, thus favouring binding in situations like this where one might not otherwise expect it. An analysis of the change in protein-bound water[9] between haemoglobin and the haemoglobin–xenon complex by a microwave technique showed an increase of protein-bound water, due to the presence of xenon. Any attempts to demonstrate this directly by X-ray methods must also await the final analysis of haemoglobin at high resolution, but changes in the charge distribution caused by xenon atoms located close to the surface of the molecule could account for the increase in bound water.

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POLYMORPHISM AND MITOCHONDRIAL ACTIVITY IN SLEEPING SICKNESS TRYPANOSOMES

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THE trypanosomes of human sleeping sickness, *Trypanosoma gambiense* and *T. rhodesiense*, are at all times morphologically indistinguishable from *T. brucei* of wild game and domestic animals in Africa, and can be regarded as genetic variants of *T. brucei* with the ability to infect man. These flagellates, previously designated the *T. brucei* sub-group, and now assigned to the subgenus *Trypanozoon* by Hoare[1], differ from other tsetse-borne trypanosomes in two important features: (1) They undergo a complex life-cycle, involving changes in form (Fig. 1), in the tsetse fly (*Glossina* spp.), developing first in the midgut and later in the salivary glands. (2) In the bloodstream of the natural mammalian host they show a wide variation in form, ranging from long slender flagellates with a free flagellum at the anterior end, to short stumpy forms with no free flagellum (Fig. 1).

Because of this second character these trypanosomes are often referred to as the 'polymorphic' trypanosomes, but this description is misleading. The morphological variation is continuous, biometrical investigations[2] giving no support to the idea that several distinct forms co-exist in the bloodstream, and any implication of a genetic basis to this multiplicity of forms was ruled out by Oehler[3], who showed that polymorphism can persist in clone infections. Strictly speaking, the flagellates are pleomorphic, but the change in form appears to follow a regular pattern in the course of infection, for the bulk of available evidence shows that slender forms are most abundant when the parasitaemia is rising, whereas short, stumpy forms preponderate when the number of flagellates in the blood is falling[3-6]; slender forms appear to transform into stumpy forms[7,8].

The cause and adaptive significance of polymorphism in *Trypanosoma brucei*-like trypanosomes are obscure. It has been suggested that the host's immune response stimulates the transformation from slender to stumpy forms[7,8]. In chronic infections the number of trypanosomes in the blood fluctuates, and it appears that each peak of parasitaemia represents a different antigenic variant[9], the trypanosomes changing their antigens to avoid the host's immune response. If host antibodies do induce the

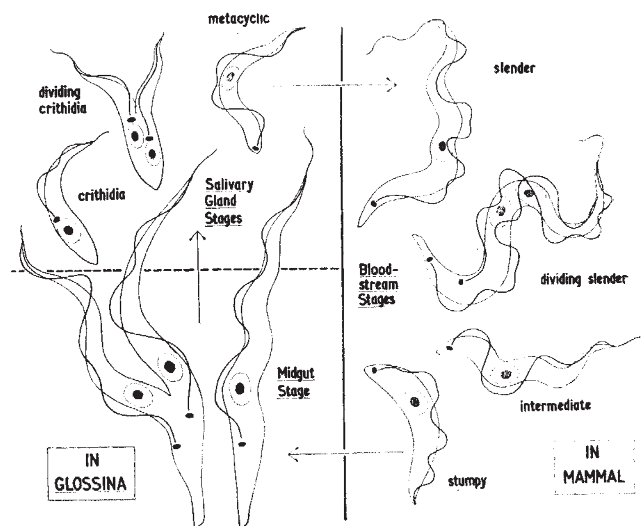

Fig. 1. Diagram of life-cycle in *Trypanosoma brucei*-like trypanosomes. In the mammalian bloodstream slender forms divide or transform into stumpy forms via intermediate forms. Stumpy forms, on entering the tsetse fly, transform into elongate midgut flagellates which, after multiplication and migration in the fly, multiply as crithidial forms in the salivary glands. The metacyclic forms which arise from crithidias are infective to the mammalian host when injected with the fly's saliva

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morphological transformation, then the stumpy forms may represent the forms which are changing their antigens; but as yet there is no direct evidence that this is so. Slender forms may be present at all stages in the infection, and Ashcroft has suggested that these forms might undergo antigenic change to give rise to the relapse strain[7].

Robertson[5], and later Reichenow[10] and Wijers and Willott[11], observed that, should infected blood be ingested by a tsetse fly, it is the stumpy, rather than the slender, trypanosome which develops in the midgut to initiate the cycle in the fly. Corroborating evidence for this comes from observations on the behaviour of polymorphic trypanosomes maintained by syringe passage in laboratory animals. After several passages the trypanosomes become monomorphic, slender-like forms only being found in the blood[12,13]. This loss of polymorphism is accompanied by loss of transmissibility by tsetse flies[13]. *Trypanosoma evansi* represents the natural counterpart of these 'non-transmissible' strains. It is morphologically indistinguishable from monomorphic *T. brucei*, and Hoare[14] has suggested that it arises from *T. brucei* when this flagellate is carried outside the tsetse belt by camels, and transferred from host to host by biting flies (for example, *Tabanus*) acting like syringes.

The behaviour of bloodstream trypanosomes on entering the fly is paralleled by their behaviour when inoculated into suitable culture media. Only strains which will infect *Glossina* will establish themselves in culture on blood agar media[15], and the forms assumed in culture are morphologically and physiologically identical with those found in the fly midgut[16].

At present, then, polymorphism in *Trypanosoma brucei*-like trypanosomes appears to be associated with cyclical transmissibility, in that its loss is accompanied by failure to develop in the midgut of *Glossina* and reluctance to be cultivated *in vitro*, but the reason why these characters should be linked is not understood.

Morphogenesis in Trypanosoma brucei. In developmental terms, the life-cycle in Protozoa can be envisaged as a series of morphogenetic responses on the part of competent cells to certain environmental changes. The cellular basis of this morphological transformation from one stage to the next in the trypanosome life-cycle is not known, but ultrastructural and biochemical investigations are beginning to provide some clues. Seen with the light microscope, the morphological changes in *Trypanosoma brucei* are extremely simple. Apart from the relative changes in length and width of the body, and length of the flagellum with respect to body-length, the most noticeable changes are seen in the spatial relationships of the kinetoplast, at the base of the flagellum, to the nucleus and to either end of the flagellate's body. In bloodstream forms the kinetoplast lies close to the posterior end of the flagellate; in the fly midgut forms it lies posterior to the nucleus but some distance from the posterior extremity of the flagellate. In crithidial forms attached to the walls of the salivary gland the kinetoplast lies immediately anterior to the nucleus, while in the metacyclics which develop from them the kinetoplast is once again at the posterior end of the flagellate (Fig. 1).

I have suggested that adaptive changes in the chondriome of *Trypanosoma brucei* and allied species might be at least partly responsible for cyclical transformation[17]. Investigations with the electron microscope have shown that the kinetoplast in trypanosomes is the DNA-containing part of the chondriome. The structure at the base of the flagellum which is stained by Romanowsky techniques and gives a positive Feulgen reaction[18] is seen with the electron microscope as a fibrous disk embedded in a mitochondrion[19–21] (Fig. 2). The kinetoplast has been known to stain with Janus green *B* for some time[22]. There is mounting evidence that DNA is a widespread component of mitochondria[23]; the large amount detectable in the trypanosome kinetoplast may indicate the presence of multiple chondriome DNA units[24].

An ultrastructural comparison of monomorphic bloodstream forms of *T. brucei* with fly midgut forms (as obtained in culture) showed that the appearance of the chondriome is very different in the two[17]. In bloodstream forms a mitochondrial tube, with scarcely any cristae, pursues a sinuous course from the kinetoplast to the anterior end of the flagellate, and a similar, but very short, tube may extend to the posterior end. The midgut forms, on the other hand, have an extensive network of mitochondrial tubes at the anterior end and a prominent post-kinetoplastic chondriome, the kinetoplast in these forms lying some distance from the posterior extremity of the flagellates. Comparative respiratory investigations of these forms give meaning to this structural difference. In monomorphic bloodstream forms, glucose is respired only as far as pyruvate which is excreted, as a functional Krebs cycle appears to be wanting in these forms[25,26]; the flagellates are almost unique in that their energy is derived from aerobic glycolysis. Although large amounts of oxygen are consumed by the flagellates, terminal respiration is independent of cytochrome pigments, which are absent, and is effected by an L-α-glycerophosphate–oxidase-L-α-glycerophosphate dehydrogenase system[27–29]; Ryley[30] has recently produced evidence that this alternative terminal respiration system is housed in distinct cytoplasmic bodies outside the chondriome. The fly midgut forms, however, respire their pyruvate completely and appear to have conventional oxidative phosphorylation linked to a Krebs cycle and cytochrome carrier system[26,27,31,32].

From comparative ultrastructural and biochemical investigations it would appear that the chondriome of the bloodstream trypanosomes is inactive, as Krebs cycle enzymes and cytochrome pigments, which are mitochondrial components, are absent. In the fly midgut, on the other hand, the trypanosome chondriome appears to be very active. On the basis of such investigations I suggested that the bloodstream trypanosome, on entering the fly, produced mitochondrial enzymes and a more extensive and active chondriome to enable it to switch its pattern of respiration to a more economical one in keeping with its new surroundings. I also suggested that adaptive proliferation of the posterior chondriome in particular resulted in the change in form, the new chondriome growing out from the kinetoplast, extending the posterior end of the trypanosome. In *Trypanosoma brucei*, then, adaptive mitochondriogenesis might explain the different positions adopted by the kinetoplast, and changes in size, which are an integral part of morphogenesis. A similar conclusion was reached for the transformations observed in the kinetoplastid flagellate *Leishmania donovani* by Rudzinska, d'Alesandro and Trager[33]. It may be noted here that the microtubules, which spiral in a longitudinal direction beneath the entire pellicle of the trypanosome (Fig. 2), probably serve as a corset directing growth along the longitudinal axis of the flagellate. Microtubules are now known to be commonly associated with elongating cells[34].

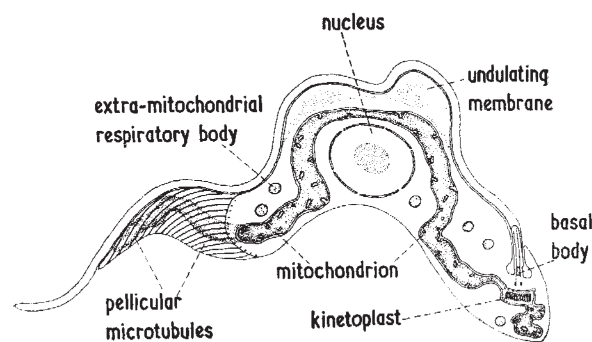

Fig. 2. Diagram of structures involved in respiration, locomotion and transformation of *Trypanosoma brucei* (intermediate bloodstream form). Pellicular microtubules are shown only at the anterior end of the flagellate

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These ideas on the transformation of bloodstream forms entering the tsetse fly, however, neglected the fact that monomorphic bloodstream forms rarely, if ever, infect the fly, whereas the stumpy forms of polymorphic strains do; an important metabolic difference between slender (or monomorphic) and stumpy forms is implied, which confers a selective advantage on the latter, enabling them to develop into midgut forms in *Glossina*.

Mitochondrial activity in bloodstream trypanosomes. Most trypanosomes do not appear to show the extremes of respiratory behaviour encountered in the monomorphic bloodstream and fly midgut forms of *Trypanosoma brucei* as recounted here. Steinert[35], using NAD diaphorase as a mitochondrial marker, has reported mitochondrial activity in both amphibian and leech (vector) forms of *Trypanosoma mega* as obtained in culture, though differences in mitochondrial morphology were evident between these stages.

After brief fixation (5 min at 4° C) in 0·1 M cacodylate-buffered 5 per cent glutaraldehyde (*p*H 7·2), I have examined the distribution of NAD diaphorase activity in several African trypanosomes and found that it agrees with the picture of the chondriome obtained from electron-microscope investigations. In bloodstream *Trypanosoma vivax* and *T. congolense* (which are not polymorphic) the single anterior mitochondrion, kinetoplast and short posterior mitochondrion are filled with blue-black formazan deposit when fixed smears are incubated for 40 min with $NADH_2$ as substrate and nitro-blue tetrazolium salt as acceptor[36,37], indicating diaphorase activity. Stages in division of the chondriome can be seen in such smears. The anterior mitochondrion appears to cleave first in the region of the nucleus, the slit extending backward to the kinetoplast and posterior mitochondrion, and forward to the extremity of the anterior mitochondrion. All the trypanosomes in a smear appear to show diaphorase activity, whereas controls incubated without substrate do not. Electron micrographs of both these bloodstream trypanosomes show well-developed cristae in the chondriome[17,38], and there is evidence[26] that pyruvate is catabolized, so that, unlike *T. brucei*, the chondriome of *T. vivax* and *T. congolense* appears to be active in the bloodstream phase.

When smears of *Trypanosoma brucei* and allied trypanosomes were tested for NAD diaphorase activity, surprising results were obtained. Old monomorphic laboratory strains showed no diaphorase activity in the mitochondria and similar results were obtained for *T. evansi*. In recently isolated polymorphic strains, however, intense enzyme activity was evident in the chondriome of intermediate and stumpy forms (Fig. 3), while slender forms resembled monomorphic forms in having virtually no diaphorase activity in the mitochondrion. It appears, therefore, that the transition from slender to stumpy forms is accompanied by the acquisition of mitochondrial activity as shown by the intramitochondrial oxidation of $NADH_2$ (Fig. 4). Controls incubated without the substrate again indicated no enzyme activity.

In slender and monomorphic *Trypanosoma brucei*, and in *T. evansi*, $NADH_2$ oxidation, as registered by formazan deposits, was occasionally witnessed in extra-mitochondrial bodies scattered throughout the cytoplasm of the bloodstream forms. These granules probably represent the location of the L-α-glycerophosphate (L-α-GP) oxidase cycle, which, as previously mentioned, mediates terminal respiration in bloodstream forms. L-α-GP dehydrogenase is NAD linked, catalysing the reduction of dihydroxyacetone phosphate to L-α-GP by $NADH_2$; L-α-GP oxidase transfers hydrogen directly from L-α-GP to atmospheric oxygen, yielding dihydroxyacetone phosphate once more.

Further evidence for activity of the chondriome in polymorphic infections comes from the ability of some of these trypanosomes to utilize Krebs cycle intermediates, particularly α-ketoglutaric acid (α-KGA) as shown by a simple motility test[50]. Ryley[30] noted that cultured midgut forms of *Trypanosoma rhodesiense* could maintain their motility if supplied with α-KGA while monomorphic bloodstream forms could not. In the work recorded here, it was found that, when bloodstream forms are washed free of exogenous respirable substrate and suspended in buffered Ringer's solution alone and with added M/10 glucose or M/10 α-KGA, motility is retained in the glucose and α-KGA long after it has ceased in the saline control (Fig. 5). Both slender and stumpy forms are active in the glucose, but in α-KGA only late intermediate and stumpy forms are active.

Morphologically, the respiratory switch is characterized by more than just a change in outward form. In many of the intermediate forms stained for NAD diaphorase activity, the mitochondrion appears to be in a state of division (Fig. 3). Steinert[39] has noted in *Trypanosoma mega* that transformation and division appear to be mutually exclusive. Of the different bloodstream forms, only slender trypanosomes are known to divide; it is possible that forms with incipient mitochondrial fission are most sensitive to the transforming stimulus. A further change noted in the transformation from slender to stumpy forms is the increase in diameter of the mitochondrial tube and the appearance of well-defined cristae within its lumen as seen with the electron microscope.

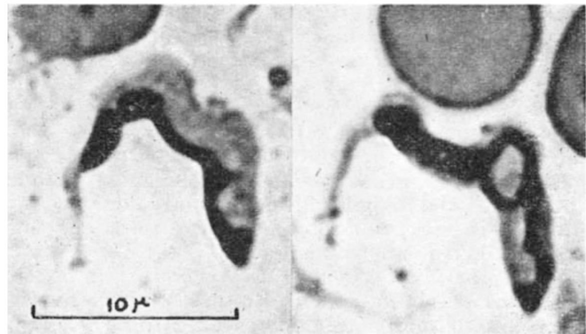

Fig. 3. Distribution of NAD diaphorase activity in late intermediate forms of *Trypanosoma brucei*. Blue-black formazan deposits indicate localization of enzyme in the mitochondrion. The mitochondrion is splitting in the trypanosome on the right

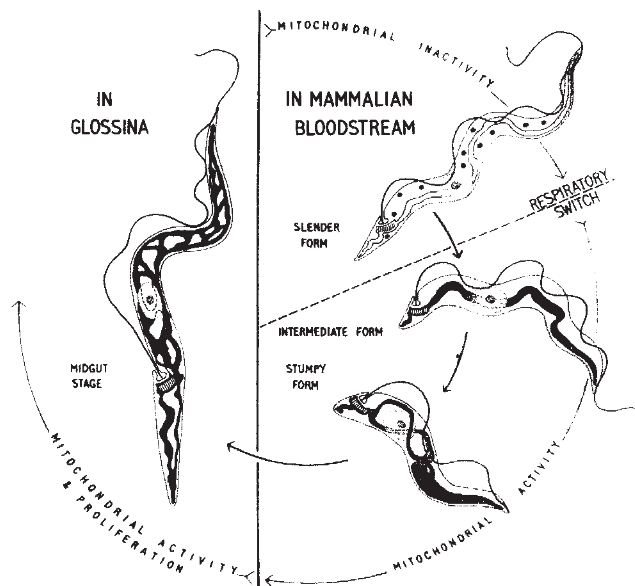

Fig. 4. $NADH_2$ oxidation by different stages in the life-cycle of *Trypanosoma brucei*. Sites of substrate oxidation are shown in black (representing formazan deposits derived from reduction of nitro-blue tetrazolium). When slender forms change into intermediate forms the mitochondrion becomes active in $NADH_2$ oxidation. When stumpy forms enter the fly, proliferation of the active mitochondrion occurs. Salivary gland stages are not shown

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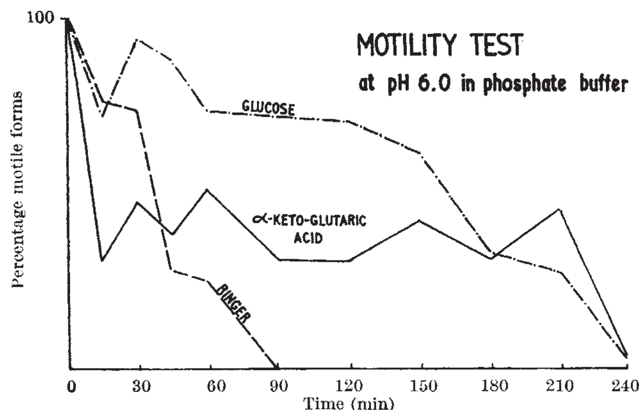

Fig. 5. Graphs to show motility of *Trypanosoma brucei* (*T. rhodesiense*) in Ringer's saline, M/10 glucose and M/10 α-ketoglutaric acid. All forms can utilize glucose to maintain motility. A fraction can also utilize α-KGA indicating mitochondrial activity. This fraction corresponds to the intermediate and stumpy forms

The nature of the stimulus which causes the transformation bringing about polymorphism and activating the mitochondrion is not known. The most likely instigator of the respiratory switch is a change in permeability of the trypanosome surface. For example, if surface permeability changed to decreased ability to take up glucose, continuation of life would be favoured by a more efficient utilization of what glucose was taken in. Host antibodies might well produce such a surface change. The shortening of body-length might be effected by a contraction of the pellicular microtubules accompanying surface change.

The assumption of mitochondrial activity by the intermediate and stumpy forms may explain why these forms survive in the fly more readily than slender trypanosomes—they are pre-adapted to the mode of respiration necessary in the midgut of the vector. The possibility that the switch might occur when slender forms enter the fly midgut cannot be precluded, however, for Wijers and Willett[11] noted transformation of slender forms to stumpy forms in the midgut of *Glossina*.

Hollingshead *et al.*[40] have noted that monomorphic bloodstream and fly midgut (culture) forms of *Trypanosoma rhodesiense* differ in their electrophoretic behaviour. It would be interesting to see if the change in surface character accompanies the slender–stumpy transformation or whether it takes place on entering the fly midgut or culture medium.

Mitochondrial activity: the genetic basis. The kinetoplast of flagellates has long been known to be a self-replicating structure (see Mühlpfordt[41] for a comprehensive review of its history). The more recent finding that it represents the DNA component of the trypanosome chondriome has brought us nearer to an understanding of its function. Steinert[20] regarded the kinetoplast as a genetic system transmitting the information necessary for the synthesis of mitochondrial enzymes. Some evidence for this is forthcoming from a study of trypanosomes which have lost the kinetoplast[42].

Monomorphic *Trypanosoma brucei*, *T. evansi* and forms closely related to them can spontaneously lose the kinetoplast as a result of faulty division and survive in the bloodstream as akinetoplastic strains[43]. The akinetoplastic condition can be induced in most trypanosomatids by treatment with certain dyes (for example, acriflavine, pararosaniline)[44], but the resulting akinetoplastic individuals are only viable in the case of bloodstream *T. brucei* and *T. evansi*, that is, those forms which do not need mitochondrial respiration in the bloodstream. The akinetoplastic trypanosomes appear to be incapable of synthesizing mitochondrial enzymes: in the work recorded here, NAD diaphorase could not be detected in akinetoplastic forms. Reichenow[45] noted the inability of akinetoplastic *T. gambiense* to infect *Glossina*. The electron microscope reveals, however, that akinetoplastic trypanosomes retain a membranous ghost of a mitochondrion ('pro-mitochondrion'), and this is found in all their progeny[42,46]. The kinetoplast may be essential for the synthesis of mitochondrial enzymes; but it is not necessary for the continuous replication of the mitochondrial membrane system.

Once lost, the kinetoplast cannot be regenerated, and this fact has enhanced its reputation of autonomy. But, as yet, there is no evidence that any cytoplasmic organelle with genetic continuity is independent of nuclear control. In the case of chloroplasts and the mitochondria of yeasts, there is ample evidence that the function of these structures is subject to the nuclear genes[24]. Reports that fusion of the kinetoplast envelope with the nuclear envelope can be observed with the electron microscope[41,42] have not yet been satisfactorily confirmed; I have never seen such fusion in examining hundreds of sections of juxtanuclear kinetoplasts from different species.

Using yeast as a model, Gibor and Granick[24] suggest that mitochondrial DNA represents a multigenic hereditary system. They envisage that, in addition to constitutive genes governing replication, other mitochondrial genes might control the differentiation of enzyme systems through operon-regulator mechanisms[47] subject to external inducers as well as nuclear influences. In anaerobically grown yeast, oxygen appears to act as an inducer to the synthesis of components of the mitochondrial respiratory system, the cytochrome chain and Krebs cycle enzymes being formed simultaneously[48]. Similar co-ordinate synthesis of respiratory enzymes can be induced by oxygen in anaerobic plague bacillus, *Pasteurella pestis*[49]. In *Trypanosoma brucei* co-ordinate synthesis of mitochondrial components appears to occur in the transition from slender to intermediate bloodstream forms, but here it is unlikely that oxygen is the inducer; the respiratory switch is not from anaerobic to aerobic respiration, but from aerobic glycolysis to complete oxidation.

The reverse respiratory switch in *Trypanosoma brucei* (that is, back to mitochondrial inactivity) probably takes place in the salivary glands of the tsetse fly, possibly during the genesis of metacyclic forms, but it could equally well occur on entering the bloodstream. The agents inducing repression of mitochondrial activity are not known.

Loss of polymorphism: evolutionary aspects. When a polymorphic strain of *Trypanosoma brucei* becomes monomorphic and non-transmissible, it presumably loses the ability to produce certain mitochondrial enzymes, for it can no longer perform the respiratory switch. The absence of selection for the respiratory switch over several generations in the mammalian bloodstream (possibly provided by rapid passage through laboratory animals) would allow the accumulation of mutations affecting the mitochondrial enzyme systems: a high rate of spontaneous mutation appears to be characteristic of cytoplasmic genetic systems[24]. A deeper analysis of the mechanisms involved in the evolution of monomorphism is scarcely possible in our present state of uncertainty about the relationship of the host's immune response to polymorphism. If we accept that, in untreated animals, antibodies produced by the host elicit both antigenic change and transformation from slender to stumpy forms in the trypanosomes[51], then we are faced with the question of whether parallel effects are registered in all flagellates or in different fractions of the parasite population. At least two possibilities present themselves.

The simplest possibility is that, under the influence of antibodies, slender forms switch their respiratory pathway and become stumpy; the antigenic change then occurs and the stumpy forms revert to slender forms, as suggested by Wijers[8]; antigenic change is closely linked with polymorphism. But this pattern of behaviour is not in keeping with the observation that syringe-passaged monomorphic strains can change their antigens without changing into stumpy forms[52]. If antigenic change is

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linked to polymorphism it is difficult to see why selection maintaining antigenic change has not also maintained polymorphism in old laboratory strains.

The elimination of polymorphism is perhaps more readily explained if antigenic change and transformation are unlinked and regarded as alternative responses on the part of the trypanosome population. Under the influence of antibodies certain slender trypanosomes might switch their respiration and transform into stumpy forms while others might change their antigens and so produce the next variant. As both transformation and antigenic variation can occur within a clone strain, the difference in response must depend on differences in competence (in the embryological sense) rather than genetical heterogeneity within the population. If antigenic change is not bound to transformation, as in this model, then it is conceivable that the ability to transform might be lost in the absence of selection, whereas the ability to undergo antigenic change might be retained, as it is in monomorphic strains. This being so, the slender forms would serve to continue the infection in the mammalian host, the stumpy forms serve to infect the tsetse fly vector. Should the stumpy forms not be ingested by a fly they are eliminated by the host.

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ANCESTRY OF SEA-STARS

In a recent series of articles, Prof. H. B. Fell[1-3] attempted to link the morphology of the skeletal elements of living sea-stars with somasteroids. This early Palaeozoic echinoderm group, originally described from the Ordovician of Europe[4], is generally held to be ancestral to the asteroids and ophiuroids of to-day. Prof. Fell's researches on living sea-stars have permitted recognition of the close comparison between the skeleton of living forms, in particular the genus *Platasterias*, and archaic somasteroids. Indeed, he refers this genus to the Somasteroidea.

This comparison of living sea-stars with the somasteroids will not be discussed here. It is a broader conclusion which invites the present comment. Prof. Fell[3] argues persuasively that somasteroids evolved from the pelmatozoan class Crinoidea, and that they are phylogenetically intermediate between later sea-stars and crinoids. Because other eleutherozoans have a shape different from sea-stars ('meridional growth gradients' in contrast to 'radial growth gradients'), seemingly these echinoderms did not evolve from crinoids. Prof. Fell therefore rejects the sub-phylum Eleutherozoa as polyphyletic, and advocates Haekel's groupings Asterozoa and Echinozoa.

Two features are taken as indicating the kinship of somasteroids and crinoids. The first is a similarity of the apical plates of many living sea-stars and the pentacrinoid calyx. The second is an apparent relationship of ossicles of a pinnulate biserial crinoid arm and of a somasteroid ray. These are discussed in turn.

(A) *The homology of the apical system of sea-stars and the crinoid calyx.* Living sea-stars, particularly ophiuroids, possess regular circlets of plates around a central adapical ossicle in their early development. These can persist to maturity so that ophiuroids may show a well-defined apical system during all growth stages (Fig. 1*c*). A similar situation may obtain in living asteroids, although the pattern is more often lost during growth, and radial elements may be absent (Fig. 1*d*). The pentacrinoid stage of development of living comatulids is very similar. There is a central plate (the dorso-central) surrounded by a circlet of five basal plates (interradial in position) and succeeded by a circlet of five radials (Fig. 1*e*). During development the basals are lost, as are small infrabasals present in some comatulids.

The various circlets of plates of these apical systems are directly homologized by Prof. Fell[3]. Now this idea is far from new. It is a partial revival of the so-called 'calycinal theory', which held sway in the latter part of the nineteenth century. As Fell neglects to discuss the facts which led to a rejection of this theory, it is pertinent to trace briefly its rise and fall.

Lovén[5] appears to have first propounded the theory in detail. He stated that the suranal plate (the central plate of the apical system of calycinid echinoids) was homologous with the centrale of the Cretaceous crinoid *Marsupites* (Fig. 1*a*, *b*) and, further, that the echinoid apical system was the homologue of the pentacrinoid calyx.

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