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GENETIC AND BIOCHEMICAL FEATURES OF THE " METHIONINE REGULATORY SYSTEM " IN *SACCHAROMYCES CEREVISIAE*

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The regulation of methionine biosynthesis in *Saccharomyces cerevisiae* is subject to a pleiotropic regulatory mechanism which involves participation of different types of macromolecules and genetic elements.

At least seven regulatory genes have been characterised: *eth1, 2, 3, 4, 10, sem4, 13.* At least two, *eth2* and *eth3*, are unlinked to each other and to the mapped structural genes *met2* (homoserine-O-transacetylase) and *met8* (homocysteine synthetase). Mutations in these regulatory loci modify simultaneously the repressibility of the two above mentioned enzymes and of two enzymes involved in the sulphate assimilation pathway (ATP sulphurylase and sulphite reductase). The product of gene ETH2 is certainly a protein since among 5 mutations obtained at this locus one is nonsense (ochre suppressible) (Masselot and de Robichon-Szulmajster, *Genetics* 1971, in press).

A thermosensitive mutation (methionyl-tRNA synthetase; locus *ts-296*) also leads to a modified repressibility with the same pleiotropic pattern as characterises the regulatory genes mentioned above. It has been shown that conditions which which promote absence of repressibility lead to a sharp decrease of *in vivo* charging of $tRNAs^{met}$. In turn, conditions which permit a re-charging of these tRNAs also lead to a return towards repressibility of met-specific enzymes (Cherest, Surdin-Kerjan and de Robichon-Szulmajster, *J. Bacteriol.*, 1971, *106*, 758), thus indicating that methionyl-tRNA (most probably a sub-species of it), rather than free methionine, is involved in this regulatory process.

The number of regulatory genes so far uncovered renders it unlikely that they are all concerned with the synthesis of an apo-repressor (even a complex heteropolymer). The hypothesis is favoured that some of these genes might be concerned either with the synthesis of a " Regulatory-$tRNA^{met}$ species ", or with the synthesis of enzymes necessary to ensure the proper composition and structure of this " Regulatory-$tRNA^{met}$ " (modified nucleosides, for example).

Some of the regulatory mutations studied, lead to considerable methionine over-production (J. Antoniewski, unpublished results), although the specific activities of the enzymes measured are not apparently " derepressed ". Massive derepression of at least one enzyme, not yet identified, has then to be postulated. Such an enzyme should be of key metabolic importance for methionine biosynthesis in *Saccharomyces cerevisiae.* Sulphate permease, APS kinase, PAPS reductase, methionine synthetase, methyl tetrahydrofolate-polyglutamyl pathway (now under study) are likely candidates for such a role.

GENETIC ELEMENTS IN THE REGULATION OF ARGININE METABOLISM IN *SACCHAROMYCES CEREVISIAE*

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The biosynthesis and the degradation of arginine in *S. cerevisiae* appear to be under the control of mutual exclusion mechanisms which involve regulatory proteins having dual functions.

1. The biosynthetic enzyme ornithine transcarbamylase (OTCase) is inhibited by arginase (epiarginase) when ornithine and arginine are present. This inhibition is the result of the reversible binding of the two enzymes (Messenguy and Wiame, *FEBS Letters*, *3*, 47, 1969).

2. At the level of gene activity it is suggested that a heteropolymeric repressor ARGR is involved in a negative type of control of the synthesis of most anabolic enzymes. The catabolic pathway appears also be under a control of a negative type exerted by a repressor (CARGR) acting on the two distinct operators linked to the structural genes for arginase and ornithine transaminase.

The effectors for repression of OTCase are all inducers of the catabolic enzymes. These effectors are arginine, ornithine and analogues including an unexpected one, lysine. The mutation *argR* (gene for ARGR) causes derepression and non repressibility of OTCase but also non-inducibility of the catabolic enzymes. It is this last and unexpected property, which has been used for the selection of operator and repressor mutations for catabolism, which overcome the incapacity to synthesise arginase and transaminase in *argR* mutants. These results strongly suggest that the ARGR repressor is ambivalent and that, when activated, it directly represses the anabolic gene and represses the synthesis or the activity of CARGR which is normally active on the two catabolic genes. In that way ARGR simulates a positive type of control (Hiernaux *et al.*, in *Current Topics in Cellular Regulation*, Vol. 4 (Ed. Horecker and Stadtman), Academic Press, 1971, and unpublished).

3. Different methods show that catabolic repression affects arginase and transaminase. This repression is largely independent of their induction. Although *argR* mutants are not inducible, they can be depressed. The comparative study of derepression of these enzymes with α-glucosidase, well known to be under carbon (energy) catabolic repression shows a behaviour clearly distinct at least from that of arginase. Growth conditions may affect α-glucosidase without affecting arginase and the converse is true.

A mutation derepressing arginase is without effect on α-glucosidase. From this, one must conclude that distinct factors affect carbon and nitrogen catabolite repression (Dubois *et al.*, in *Current Topics in Cellular Regulation*, Vol. 4 (Ed. Horecker and Stadtman), Academic Press, 1971, and unpublished).

SOME FEATURES OF THE CONTROL OF PURINE SYNTHESIS IN *SACCHAROMYCES CEREVISIAE*

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Mutants of the genes *pur 1* to *pur 6* excrete purines and are presumed to be defective in the regulation of *de novo* purine synthesis. Two of them have been shown to be allelic to genes specifying steps in the purine pathway; *pur 6* is allelic to *ade 4*, which lacks activity of the first enzyme PRPP amidotransferase, and *pur 1* is allelic to *ade 12*, which is thought to code for adenylosuccinate synthetase. Several other genotypes, *pur 6*, *su-pur*$^+$, *su-pur* and *dap* are functionally allelic to *ade 4* and are presumed to specify different forms of PRPP amidotransferase. These genotypes vary in their response to feedback inhibitors, repression by endogenous purines are resistance to purine analogues. They also differ in their capacity to carry out the alternative first step of purine synthesis (ribose-5-phosphate + ammonia → phosphoribosylamine). Activity of PRPP amidotransferase has also been studied in the other *pur* mutants; all are sensitive to feedback inhibition but insensitive to repression.

REGULATION OF ARGININE METABOLISM IN *ASPERGILLUS*

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Arginine biosynthetic and catabolic pathways represent repressible and inducible systems respectively. Most of the data on genetic regulation of these two systems in *Aspergillus nidulans* were obtained from the studies on the synthesis of three enzymes: ornithine transcarbamylase in the biosynthetic pathway, and arginase and ornithine transaminase in the catabolic pathway. The kinetics of induction or repression of these enzymes was investigated and some data concerned with the time of transcription and translation of the relevant structural genes and the half-life of the corresponding mRNA's were obtained.

By means of a positive selection technique several hundreds of mutants showing elevated levels of one or both catabolic enzymes were isolated. The preliminary genetic analysis of these mutants revealed that they map in several distinct loci (no less than ten). Mutations at two out of three loci studied more intensively affect simultaneously the synthesis of biosynthetic enzymes. This provides evidence for the common genetic regulation of biosynthetic and catabolic pathways. Non-inducible mutants for catabolic enzymes were also obtained.

THE CONTROL OF NITROGEN METABOLISM IN *ASPERGILLUS NIDULANS*

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In *Aspergillus*, ammonium, by repression and/or inhibition, prevents the catabolism of many nitrogenous compounds. To study whether nitrate exerts a similar effect, strains mutant in the production of nitrate reductase (*nia* and *cnx*), of nitrite reductase (*nii*), and of both these enzymes (*nir*$^-$) have been tested for growth on various nitrogenous compounds in the presence and absence of nitrate. The results obtained are complex. Some, but not all, nitrate reductaseless strains show a reduced ability, *vis á vis* wild type, *nii* and *nir*$^-$ strains, to utilise certain amino-acids as nitrogen source. In the presence of nitrate a similar pattern is obtained, except that *nii* strains grow much less, showing a nitrogen starved rather than an inhibited morphology. *nir*$^-$ mutations are epistatic to *nia* and *nii* mutations are epistatic to *nii*. Thus it appears that nitrate reductase must be synthesised if certain amino-acids are to be catabolised, but only provided the *nir* product is present. Nitrate prevents this catabolism, but by way of nitrate reductase and the *nir* product.

An alternative approach has been the study of chlorate toxicity. The patterns of toxicity obtained are consistent with chlorate mimicking nitrate's inhibiting effect on the catabolism of nitrogenous compounds. Again nitrate reductase and *nir* product are necessary for the mediation of this effect. Mutants have been obtained which are resistant to chlorate, but retain their ability to grow on nitrate. The biochemical and genetic bases of these are being investigated and this should shed further light on the mechanism of chlorate toxicity.

Meanwhile it is tentatively proposed that the *nir* product and nitrate reductase play both a positive and negative role in regulating the catabolism of some nitrogenous compounds.

REGULATION OF NITRATE ASSIMILATION IN *NEUROSPORA CRASSA*

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Assimilation of nitrate by *N. crassa* is affected by mutation in any one of at least five genes. Growth tests using nitrate, nitrite, ammonium, hypoxanthine and uric

acid as sole nitrogen sources indicate that two of these genes *nit-1* and *nit-3* are concerned with the synthesis of nitrate reductase. Biochemical evidence has confirmed that mutants at both these loci possess wild type levels of nitrite reductase. Mutants at two other loci, *nit-4* and *nit-5* appear to be fully repressed, under all growth conditions tested, for both nitrate reductase and nitrite reductase. Enzyme levels in these mutants are identical to wild type when grown on ammonium ion as sole nitrogen source i.e. they produce small but measurable amounts of both enzymes. Mutants at the fifth locus *nit-2* appear to be completely lacking in both activities. This and other evidence suggests that mutation at this locus prevents the utilisation of a wide variety of nitrogen sources including nitrate. Induction experiments involving wild type and *nit-1* and *nit-3* mutants suggest that in the wild type both enzyme activities are positively controlled by nitrate and nitrite ions. In the mutants, however, both nitrite reductase and activities associated with nitrate reductase (reduced benzyl viologen nitrate reductase in the case of *nit-3* and NADPH-cytochrome c reductase in the case of *nit-1*), are de-repressed by the removal of ammonium ions. In the case of nitrite reductase, however, the level of this enzyme is enhanced by the presence of nitrate ion indicating that two control systems could be operating simultaneously.

AMMONIUM REPRESSION AND THE REGULATION OF AMMONIA TRANSPORT IN *ASPERGILLUS NIDULANS*

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In *Aspergillus nidulans* the synthesis of the following enzymes or uptake systems is repressed by ammonia: nitrate reductase; hypoxanthine dehydrogenase; extracellular protease; urea uptake; glutamic acid uptake. Mutation in at least five loci can result in ammonia derepression for some or all of these systems. In addition, mutation in four and possibly also in the fifth of these loci can result in abnormalities of ammonia transport. The properties of these mutants will be described. The complex relationships between the derepression and transport phenomena are most easily explained by the postulate that the products of one or more of the known genes have both a regulatory and transport function.

CONTROL OF XDH I AND XDH II IN *ASPERGILLUS NIDULANS*

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There are two xanthine dehydrogenases (XDH) in *Aspergillus nidulans*. XDH I is induced by uric acid, XDH II is constitutive in *apl*^{r} mutants, can be induced by nicotinic acid and has weak nicotinate dehydrogenase activity.

The two enzymes share the *cnx*-determined molybdenum-containing cofactor and the polypeptide coded by the *hxB* locus. The polypeptide coded by the *hxA* locus is specific for XDH I and carries most probably the substrate binding site. Recent evidence indicates that XDH II also contains a specific polypeptide.

XDH I and urate oxidase are under the control of the *uaY* locus, at which both pleiotropically negative and partially constitutive mutations are known.

XDH II is controlled independently from urate oxidase. The differential induction indicates that two related but different enzymes can be produced by modifying a common core (coded by *cnx* and *hxB*) with a specific peptide. It is not yet clear whether the common core is constitutive or if it can be induced by both urate and nicotinate. There is some evidence (for *hxB*) favouring the second possibility. Both XDH I and II are subject to ammonia repression. XDH II is also strongly repressed by nitrate; data presented elsewhere (Scazzocchio and Holl, *Biochem. J.*, in press) indicates that the aporepressor is the nitrate reductase protein.

THE EFFECT OF NITRATE ON THE ACTIVITY OF THE D-MANNITOL-1-PHOSPHATE DEHYDROGENASE OF *ASPERGILLUS NIDULANS*

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Several *Aspergillus* species have been shown both to accumulate D-mannitol from glucose (Lee, *Appl. Microbiol. 15*, 1206, 1967) and to utilise it as sole carbon source. Mannitol-1-phosphate dehydrogenase which catalyzes the interconversion of D-fructose-6-phosphate and D-mannitol-1-phosphate, may be involved in the synthesis of D-mannitol, and perhaps also in its catabolism (Lee, *Biochem. Biophys. Res. Commun. 29*, 337, 1967 and Strandberg, *J. Bacteriol. 97*, 1305, 1969).

Growth of *Aspergillus nidulans* in glucose medium with urea as sole nitrogen source, leads to ten-fold higher activity of mannitol-1-phosphate dehydrogenase than growth with urea and sodium nitrate. The characteristics of mutants of the nitrate reduction pathway (Cove, *Proc. Roy. Soc. Lond. B. 176*, 267, 1970) suggest that neither nitrate, nor the *nir* product active with respect to the induction of nitrate reductase, is the immediate effector, but that the nitrate reductase molecule bound with nitrate may be.

A SECOND METHYLAMMONIUM RESISTANCE LOCUS IN *ASPERGILLUS NIDULANS*

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Among the methylammonium resistance mutations previously described (Arst, and Cove, *J. Bacteriol. 98*, 1284, 1969) are alleles at a minimum of two loci, but mutations at both loci lead to derepression in the presence of ammonium of a large number of ammonium-repressible syntheses. These mutations have been compared with respect to level of resistance conferred, dominance relationships, pleiotropic effects, and map positions in the genome, and the properties of double mutants carrying mutations at both loci have been investigated.

THE ARGININE PATHWAY IN *NEUROSPORA*: ENZYMES, POOLS AND FLUXES IN HETEROKARYONS

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Arg 12, *arg 1*, and *arg 10* are the structural loci for the enzymes ornithine transcarbamylase, argininosuccinate synthetase and argininosuccinase respectively. Three types of heterokaryons were constructed containing, as forcing markers, *ad 4*$^-$ and *arg 12*$^-$ or *arg 1*$^-$ or *arg 10*$^-$. For each type, heterokaryons with a range of nuclear ratios (*ad 4*$^-$/*arg*$^-$) were produced and the activities of the three enzymes determined. The relationship between nuclear dose and enzyme activity is almost linear but there is some derepression particularly at low doses.

The flux of arginine " out " of the pathway to protein and urea was measured as was the growth rate for all the strains. Reduction in flux and growth occurs only at low levels of arginine. The flux through steps prior to the reduced enzyme may increase and this is reflected in an increase in the steady state pool sizes. The increased flux can be accounted for by derepression of enzymes early in the pathway.

THE ARGININE PATHWAY IN *NEUROSPORA*: ENZYME REGULATION IN REVERTANTS

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Induced revertants from *arg 12*$^-$ or *arg 1*$^-$ or *arg 10*$^-$ can be obtained with greatly reduced specific activities for transcarbamylase, synthetase and argininosuccinase respectively. The genetic substitution of any one wild-type allele by a revertant or the combination of several of them generated a set of strains with reduced extractable arginine pools when grown under exponential growth conditions. The lowest pool concentration achieved was 2% of the wild-type level. All strains are prototrophic and grow on minimum medium.

If attention is focused on the enzymes controlled by the wild-type alleles in the presence of varying " internally " generated arginine pools, the activity of the genes can be assessed. Increases in enzyme activities can be correlated with decreases in the arginine pool. As all assays were carried out with dialysed extracts, inhibition is excluded and the phenomenon is attributed to enzyme derepression. All three enzymes exhibit derepression with decreasing arginine pool but to different degrees. Arginine supplementation of the medium, resulting in internal arginine levels about 6 times that of wild-type, represses all activities to the same level which is slightly elevated above that of wild-type when grown on minimal medium.

FLUX REGULATION IN ENZYME PATHWAYS

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The movements of enzymes, pools and fluxes consequent upon genetic changes are complex and raise difficulties in interpretation.

A theoretical treatment of enzyme systems and their response to changes in enzymic parameters has been shown to go towards the understanding of the problem.

DIVERGENT TRANSCRIPTION OF THE *argECBH* CLUSTER IN *ESCHERICHIA COLI*

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The *E* and *CHB* genes of the *E. coli* K12 arginine cluster are transcribed on to separate mRNA molecules (Cunin *et al.*, *M.G.G. 106*, 32, 1969; Baumberg & Ashcroft, *J. gen. Microbiol.*, in press), the latter group from *C* through *H*. Elseviers *et al.* (*FEBS Letters, 3*, 18, 1969) described strains carrying deletion *sup 102* which covers all mutations in *C* and *B* but none in *E* or *H*. These strains showed no E enzyme activity *in vitro*, though slight activity *in vivo*: H activity was less repressible than in wild-type, resembling rather wild-type E activity. They suggested that (*a*) *sup 102* crosses the *E-C* boundary, but leaves *H* intact, and (*b*) *E* is transcribed in the same direction as *CBH*, *sup 102* placing *H* under the control of a promoter-operator region to the left of *E*.

We have selected derivatives with restored E activity from a *sup 102*-carrying strain. Most of the isolates differ from wild-type in maximal activities of the E and H enzymes and/or in their repressibility; some completely lack H activity, in certain cases through delection of part or all of the *H* gene. We interpret the results to indicate that (i) *sup 102* leaves *E* intact, and (ii) transcription of both *E* and *CBH*

occurs outwards from a common promoter-operator region at the *E-C* boundary. G. A. Jacoby (personal communication) has recently reached the latter conclusion independently.

REGULATION OF SEX PILUS FORMATION BY RESISTANCE FACTORS IN *PROTEUS MIRABILIS*

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Genes on a sex factor within a *Proteus mirabilis* host often show patterns of expression different from those in other enterobacterial hosts (Colby & Hu, *Biochim. biophys. Acta 157*, 149, 1968; Dale & Smith, *Biochem. J. 123*, 493, 1971); another facet thereof may be relaxed control of sex factor replication in this organism. We have compared the proportion of cells bearing sex pili in cultures of *E. coli* and *P. mirabilis* containing F-like resistance factor R1, which shows normal self-repression of piliation, or depressed mutants R1-16 (repressor insensitive, i^+o^c) and R1-19 (repressor-deficient, i^-o^+) derived from it. Piliated cells were estimated as those which adsorbed male-specific phage MS2.

The percentages of MS2-adsorbing cells in actively-growing *E. coli* cultures carrying R1, R1-16 and R1-19 were 0·22, 66 and 33 respectively, while the corresponding figures for the R factor-carrying *P. mirabilis* cultures were 1, 0·8 and 0·9. This system, therefore, shows similarities—diminished maximal level of gene expression and contracted range between repression and derepression in *P. mirabilis* as compared to *E. coli*—to the *E. coli* lactose system as reported by Colby & Hu. The fraction of piliated cells declines throughout the stationary phase in both *E. coli* and *P. mirabilis* carrying R1. A comparison of these results with determinations of frequency of R factor transfer from the various *E. coli* and *P. mirabilis* strains will be made.

PHAGES T2 AND T4 DO NOT PRODUCE HEAT STABLE DENSITY MUTANTS: A TEST OF THE HEADFUL HYPOTHESIS

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The headful hypothesis (Streisinger *et al.*, *P.N.A.S. 57*, 292) describes certain features of the maturation of chromosomes of phages containing DNA with permuted base sequences, e.g. T2 and T4. Phage sized lengths are cut from longer intracellular DNA molecules (concatemers) by a mechanism which removes fixed lengths or headfuls of DNA apparently regardless of sequence. The deletion of inessential regions from the DNA would not influence this mechanism, the deleted sequences being compensated for by the chromosome having a longer terminal redundancy. Some evidence is available in support of this hypothesis. By contrast it is believed that chromosomes of phages having DNA molecules with non-permuted sequences (e.g. the T-odd phages) are excised from concatemers by a mechanism which specifically recognises the ends of viral chromosomes. Therefore, viable particles containing deletion mutations would have shorter DNA molecules and be less dense. Such mutants have been found. The mutant particles are characteristically more heat stable than their wild types. The T-even phages would not be expected to produce such heat stable mutants. Experiments designed to isolate heat stable mutants from T2 and T4 have failed to show their existence and this result is taken as further support for the headful hypothesis.

A PLASMID ASSOCIATED WITH FERTILITY AND MORPHOLOGICAL DIFFERENTIATION IN *STREPTOMYCES COELICOLOR*

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Wild-type *Streptomyces coelicolor* A3(2) is of the IF (Initial Fertility) type. NF (Normal Fertility) strains are all derived by recombination from a single NF strain in the pedigree of strains descended from A3(2). The change from IF to NF has not been repeated and is presumed to be due to a rare event, possibly a conventional mutation. The NF/IF difference has been located at the 9 o'clock region of the chromosome in NF × IF crosses (Vivian & Hopwood, *Journal of General Microbiology, 64*, 101, 1970).

IF strains give rise to UF (Ultra-fertile) variants, which show a greatly enhanced fertility when crossed with NF strains (Hopwood, Harold, Vivian & Ferguson, *Genetics, 62*, 461, 1969). Both IF and UF strains behave as recipients when crossed with NF strains, which behave as donors.

IF × UF crosses yield recombinants for chromosomal markers with a frequency of only 10^{-5} per total progeny, while members of the UF parental genotype are virtually all converted from UF to IF. The conversion from UF to IF is thus due to the acquisition of an extrachromosomal element, a plasmid, designated SCP1 (Vivian, *Journal of General Microbiology, 69*, in press, 1971). Conversely the production of UF strains from an IF culture involves loss of the plasmid; this is compatible with the high rate of spontaneous origin of UF from IF strains.

Some experiments will be described to show that efficient conversion of UF to IF in IF × UF crosses involves the production of a diffusible substance by IF strains which specifically inhibits the development of aerial mycelium by UF strains. The implications of this association between a fertility factor and morphological differentiation will be discussed.

ExrA MUTANTS OF *E. COLI*: MUTABILITY AND ABILITY TO REPAIR GAMMA-RAY INDUCED SINGLE STRAND DNA BREAKS

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ExrA (w) strains were originally derived by nitrosoguanidine mutation from radiation sensitive strains B_{s-1} and B_{s-2} and were characterised by their lack of mutational response to ultraviolet light (Witkin, *Proc. Symp.* "*Recovery and repair mechanisms in radiobiology*", *Brookhaven N. L.*, 1967) and ionising radiation (Bridges, Law & Munson, *Molec. Gen. Genet.* 103, 266, 1968). ExrA (w) strains have been found to be no more sensitive to ionising radiation than ExrA $(w)^+$ mutants (characterised by UV and γ-ray mutability), and both ExrA $(w)^+$ strains are equally good at repairing single-strand DNA breaks as revealed by alkaline sucrose centrifugation. When the *exrA* allele was introduced into wild-type strains by transduction, the resulting bacteria were non-mutable by UV and γ-radiation, were as sensitive to γ-radiation as *recA* bacteria, and were totally unable to repair single-strand DNA breaks in growth conditions. The results show that the phenotype of the *ExrA* $(w)^-$ strains cannot be attributed to the *exrA* allele. They also show that it is possible for the DNA single-strand breaks detected under our experimental conditions to be repaired accurately (i.e., without induction of mutations). The mutagenic potential of single-strand breaks is thus only realised in conjunction with certain repair functions.

SPERMINE ANTIMUTAGENESIS IN *E. COLI*

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Spermine, at a concentration of 100 μg/ml, has been tested for antimutagenic activity in a series of $trp^- \longrightarrow Trp^+$ reversion systems in *E. coli* B/r and K-12. The

Trp+ reversions scored have been of spontaneous origin and induced by 2-aminopurine during replication. The Trp+ reversions could be sub-divided, in the case of ochre *trp* mutants, into presumed true back-mutations and suppressor mutations by their ability to support the lytic growth of phage T_4 ochre mutant C428. In the case of tryptophan synthetase Trp+ revertants these could also be subdivided on the basis of differential sensitivity to 5-methyltryptophan inhibition and accumulation of indole glycerol (Yanofsky, Ito and Horn, *Cold Spr. Harb. Symp.* 31, 151, 1966). These tests allow one to determine whether or not spermine antimutagenesis is specific for any sub-class of Trp+ reversions, and hence for any particular type of basepair substitution. Furthermore, the use of a pair of excision repair competent and deficient strains carrying the same *trp*− marker allowed one to test for an influence of excision repair on 2-aminopurine mutagenesis and/or spermine antimutagenesis.

ULTRAVIOLET LIGHT SENSITIVE MUTANTS OF *COPRINUS LAGOPUS*

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Four UV-sensitive mutants (S_5, S_8, S_9, and S_{10}) have been isolated from wild type *Coprinus lagopus* BC9/66 using a new replica plating technique (loop replica plating method). The mutants S_8 and S_9 are more sensitive to UV light than S_5 and S_{10}.

Complementation tests suggest that there are at least three genes, each of which may mutate to UV sensitivity. Mutants S_9 and S_{10} apparently do not complement in a dikaryon, but all other pairwise combinations give dikaryons as resistant or more so than wild type dikaryons.

The mutant strain S_5 shows no photoreactivation and may therefore be deficient in photorepair. On the other hand strains S_8, S_9 or S_{10} show photoreactivation.

EFFECT OF LIGHT ON VIABILITY OF *COPRINUS LAGOPUS*

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It was noticed that oidia of *Coprinus lagopus* incubated in light showed a lower viability than those incubated in dark. Continuous illumination for 24 hours under fluorescent light reduced viability of oidia of the wild type strain BC9/66 to 0-10% of control in dark, but not all strains tested are equally sensitive. Further investigation has provided evidence that the wavelengths effective are in the blue and near UV regions. The temperature of incubation affects the degree of sensitivity in oidial suspension.

Preliminary experimental results suggest that light sensitivity is under polygenic control.

PHOTO-REACTIVATION AND PHOTO-PROTECTION OF ULTRA-VIOLET RADIATION DAMAGE IN CONIDIA OF *ASPERGILLUS NIDULANS*

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Photo-reactivation of ultra-violet damage in conidia of *Aspergillus nidulans* has recently been demonstrated (Fortuin, *Mut. Res.*, *13*, 131). He reported that no increase in survival occurred if conidia were treated with photo-reactivating light prior to the ultra-violet irradiation (photo-protection). Results will be presented which confirm the occurrence of photo-reactivation, but which also provide evidence for photo-protection. Dose response curves showed that a plateau for photo-reactivation was reached after about 60 minutes exposure to photo-reactivating light.

At 0·1% ultra-violet survival levels, a 20-fold increase in survival was obtained. About half of this increase could be accounted for by photo-protection.

The level of survival normally used in mutagenic experiments is 1% where a 10-fold increase in survival was observed. It is apparent that precautions should be taken to reduce photo-reactivating light before and after ultra-violet treatment if reproducible results are to be obtained.

HYPHAL FUSIONS IN RELATION TO INCOMPATIBILITY IN *COPRINUS LAGOPUS SENSU* BULLER

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Sexual morphogenesis in *Coprinus lagopus* and in the related tetrapolar basidiomycete, *Schizophyllum commune*, is subject to genetic regulation by the two independent *A* and *B* incompatibility factors. The evidence to date has not suggested any marked differences between the morphogenetic sequences in the two organisms.

Ahmad and Miles (*Genet. Res., Camb. 15*, 19, 1970) have demonstrated that there exists in *S. commune* a relationship between mating-type and the frequency of hyphal fusions, and that fusion frequency is independent of temperature. *A*-factor heterozygosity was shown to be necessary for high fusion frequency.

In a comparable analysis using *C. lagopus* a relationship between mating-type and fusion frequency was also found to exist. However, the temperature dependence of fusion frequency suggested enzymatic control. *B*-factor homozygosity was found to depress fusion frequency and *A*-factor homozygosity almost certainly has the same effect. An additional feature was the relatively high fusion frequency in non-compatible matings.

A GENETIC STUDY, INCLUDING EVIDENCE FOR HETEROSIS, OF CYSTIC FIBROSIS OF THE PANCREAS

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Autosomal recessive inheritance of cystic fibrosis of the pancreas is well established. This disease is the commonest human single gene defect in Northern Europe and N. America with an incidence of about 1 in 2,000, and it shows marked phenotypic variation. It has been suggested that both the high incidence and the phenotypic variation may be due to heterogeneity or to multiple allelism. The alternative hypothesis of heterosis is supported by the finding by previous groups of workers of increased fertility of heterozygotes. However, the observed increase, which is in excess of that required to maintain the high gene frequency, may be due to control bias.

In the study reported here, a family survey excludes heterogeneity or multiple allelism for the great majority of cases, although there is evidence that a small minority of atypical cases may be due to a different gene. Phenotypic variation, not clearly due to differences in treatment, would appear to be due largely to modifying genes. Using the cilia test no evidence of linkage was found to any one of several marker genes. A study of differential morbidity showed a significantly lower incidence of tuberculosis among index parents compared with control parents.

PROGRESS IN THE IDENTIFICATION OF THE CILIA-FACTOR PRESENT IN THE PLASMA OF PATIENTS WITH CYSTIC FIBROSIS

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Plasma of children affected with cystic fibrosis (an autosomal recessive disorder) contain a factor which inhibits ciliary beat of the gills of the fresh-water mussel *Dreissensia polymorpha.* A similar effect is seen with plasma from the parents of these children (i.e. heterozygotes). Attempts are being made to isolate and characterise this factor.

PARTIAL LINKAGE BETWEEN HUMAN GENES IN MAN-MOUSE CELL HYBRIDS

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Cell hybrids between man and other species, with subsequent large-scale loss of human chromosomes, would seem to provide ideal material for the recognition of synteny (location of the same chromosome) between human genes. Yet the results so far have been disappointing.

Ruddle *et al.* (*Nature, New Biol. 232*, 69, 1971) monitored 64 clones from man-mouse hybrids for the presence of 17 human enzymes, and apart from three which were known already to be sex-linked, no synteny was found. There was, however, strong evidence of non-random segregation of the 14 enzymes. This evidence will be presented and its explanation will be discussed.

ROBERTSONIAN TRANSLOCATION AND CHROMOSOME POLYMORPHISM IN MAMMALS

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Robertsonian translocation (" fusion " of two acrocentric chromosomes to form one metacentric chromosome) is a common type of chromosome mutation in mammales. The frequency in man may lie between one in five thousand and one in ten thousand gametes. Presumptively balanced polymorphic systems based on Robertsonian translocation are widespread, a notable example being that in the common shrew. Segration at first meiotic anaphase should be regular in both homozygotes. In heterozygotes, convergent orientation of the trivalent and regular segregation could also be expected, but irregular segration, leading to the formation of unbalanced gametes, may take place from time to time and thereby confer a selective disadvantage on the heterozygote. Limited information obtained from study of a shrew population suggests that the system could nevertheless be maintained by overall heterozygote advantage. Observations of the segregational properties of single Robertsonian chromosomes in the mouse and parallel tests of fertility have now shown that the output of unbalanced gametes may differ considerably between animals with similar metacentric chromosomes, but heterozygous for different translocations. The results do not show whether the specific frequency is wholly an expression of inherent properties of the chromosome concerned, or whether it is influenced by interaction with the residual genotype. In either case the observations present an unexpected problem for chromosome mechanics and karyotype evolution.

A STUDY OF LINKAGE DISEQUILIBRIUM IN *DROSOPHILA MELANOGASTER* POPULATIONS

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We carried out an experiment to detect possible linkage disequilibrium between five polymorphic loci (controlling enzymes) which are located in the middle of chromosome 3 of *Drosophila melanogaster*. Sets of chromosomes were extracted from male flies by a balancer technique and maintained either homozygous or as balanced stocks. Electrophoresis using starch and acrylamide gels was employed to score each extracted chromosome for its genotype with respect to the five loci. We looked at three sets of chromosomes: two were samples from successive years of the same natural population (Ives' Amherst, Massachusetts population) and one was a sample from an artificial population which had been maintained in the laboratory for several years. Most pairs of genes showed no evidence for linkage disequilibrium, but statistically significant effects were detected in a small number of cases. There was no evidence for the segregation of chromosome 3 inversions in these populations.

THE MAINTENANCE OF LIFE-CYCLE VARIABILITY IN *MYZUS PERSICAE*

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In temperate climates some aphids have a variable life-cycle, either producing sexual forms in the autumn in response to decreased daylength, or continuing parthenogenetic reproduction through the winter. Sexual reproduction results in a cold-resistant overwintering egg, whereas the overwintering success of those aphids which continue parthenogenesis is dependent on the severity of the winter. In *Myzus persicae* it is common for the continuously parthenogenetic forms to produce some males in autumn, and thus contribute to the sexual phase. Breeding experiments suggest that these males are part of a genetic system by which the alternative methods of overwintering are maintained from one year to the next. The life-cycle differences appear to be simply inherited, with the continuation of parthenogenesis through winter perhaps due to the operation of a recessive switch-gene.

REGULATION OF GLUTAMATE TRANSPORT IN *ASPERGILLUS NIDULANS*

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Aspergillus nidulans has a high affinity transport system for glutamate and aspartate which is energy dependent and has a Km of about 2×10^{-4}M. The system can transport glutamate or aspartate against a concentration gradient. In spite of glutamate utilisation, wild type cells can concentrate free L-glutamate 25-50 fold compared with the exterior concentration. The synthesis of the glutamate uptake system is repressed by ammonia. A number of mutants which are derepressed for certain other ammonium-repressed uptake and enzyme systems are also derepressed for glutamate uptake.

A mutant which has lost the activity of the glutamate uptake system has been obtained. Studies on amino acid uptake systems in this mutant as well as competitive inhibition studies with the wild type strain have been done. These show that the glutamate and aspartate uptake system is specific for these amino acids.

Two classes of mutants which are impaired in the uptake of other neutral and acidic amino acids as well as glutamate and aspartate have been isolated. One class of mutants is recessive, the other dominant, in the heterozygous diploid.

UREA AND THIOUREA UPTAKE IN *ASPERGILLUS NIDULANS*

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It will be shown that urea uptake in *Aspergillus nidulans* is a specific, saturable and energy requiring system, and that this system is repressed by ammonia. Some of the mutants which are derepressed for other ammonia repressible systems, such as nitrate reductase and glutamic acid uptake etc., are also ammonia derepressed for urea uptake.

Thiourea is an analogue of urea which we have found to be toxic to wild type *Aspergillus nidulans*, and which is taken up by the same system as urea. Thiourea resistant mutants have been isolated which are abnormal with respect to urea and thiourea uptake. These mutants are semi-dominant in the heterozygous diploids.

It is postulated that thiourea resistance is at least partially due to the reduced rate of thiourea uptake in these mutants.

LACTOSE UTILISATION AND β-GALACTOSIDASE ACTIVITY IN *ASPERGILLUS NIDULANS*

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β-galactosidase activity in *A. nidulans*, defined by the ability to hydrolyse O-nitrophenyl-β-D-galactoside, is inducible. The specific activity of an extract from lactose-grown or galactose-grown mycelium is more than 50 times higher than that from mycelium grown on glucose, sucrose or glycerol.

Gel chromatography and sucrose gradient techniques show the existence of two molecular species with β-galactosidase activity, the latter technique giving approximate molecular weights of 450,000 and 120,000. Present evidence suggests that these activities are both coded by the same gene, and represent different molecular states of the same polypeptide.

In previous studies on the system in *A. nidulans*, mutants growing poorly on lactose have been isolated, but none of these were found defective in β-galactosidase activity.

A histochemical staining technique has been developed which has made it possible to screen colonies for β-galactosidase activity directly.

Among 30 nitrosoguanidine-induced mutants selected for poor growth on lactose one, *lac*$^{-}$5, was found to lack β-galactosidase. It has been shown that the two phenotypic alterations are due to mutations at two unlinked loci. Four classes of progeny are obtained when *lac*$^{-}$5 is crossed to wild type—the two parental classes and two recombinant classes, which are:

1. Strains growing poorly on lactose, but with a normal β-galactosidase level when induced by galactose.

2. Strains growing normally on lactose, but with no detectable β-galactosidase.

Representative strains of the two recombinant classes have been intercrossed, and backcrossed to wild-type. All these crosses confirm the hypothesis of two genetic loci and show that lactose utilisation and β-galactosidase activity in *A. nidulans* are separable. Two problems are raised by this finding: firstly, the physiological significance of β-galactosidase in *A. nidulans*, and secondly, the nature of the alternate (presumably major) mode of lactose utilisation in the organism.

GENETIC CHANGES IN CONTINUOUS CULTURES OF YEAST

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Reduced temperatures induce high frequencies of petite mutants in some strains of yeast (Ogur, Ogur & St. John, *Genetics 45*, 189, 1960). In continuous culture, dramatic increases in the proportion of respiratory-deficient mutants occur at 15, 18 and 21° C. Similar results using either glycerol or glucose as carbon-sources show that this effect is due to mutation-induction and crosses show that induced petites are cytoplasmic. Other respiratory-deficient mutants, both capable and incapable of reversion to wild-type and having growth rates approximately equal to wild-type, have been detected. These are both cytoplasmic and segregational mutants.

Continuous culture selects a respiratory-sufficient line in which petites are not induced by reduced temperatures. The genetic change in cells of this line is not yet known but presumably they contain mitochondrial DNA insusceptible to low-temperature mutation. Selection of stable lines explains why commercial brewing strains show no induction of petites by reduced temperatures.

An increase in the proportion of mitotic segregants of heterozygous diploid strains is expected to occur during prolonged culture. This has been observed.

ISOLATION AND ANALYSIS OF TEMPERATURE SENSITIVE MUTATIONS AFFECTING SPORE GERMINATION AND GROWTH IN *ASPERGILLUS NIDULANS*

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Spore germination in *Aspergillus nidulans* exhibits partial synchrony (Bainbridge, *J. gen. Microbiol. 66*, 319) and this system offers a number of advantages in the study of the genetic control of developmental processes in fungi.

Mutants have been isolated following mutagenesis and filtration enrichment of spores germinated at 45°. These mutants grow well at 25°, 30° and 37°, but grow poorly or not at all at 45°. A range of phenotypes have been observed, varying from no germination or spore swelling to the production of mis-shapen spores and germ tubes. On the basis of experiments involving the transfer of plates, after germination, from 37° to 45°, the mutants can be classified into two groups. In one group of mutants, growth is temperature sensitive, whereas in the other, the germination process itself appears to be temperature sensitive.

SOME CHARACTERISTICS OF CONIDIATION MUTANTS IN *ASPERGILLUS NIDULANS*

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Conidiation mutants have been isolated after NTG treatment. The majority are oligosporogenous and variable. The degree of expression of the mutant phenotype can be altered by cross feeding from other mutants or wild types, changing carbon or nitrogen sources, temperature, pH, adding a variety of vitamins or amino acids to the medium or growing on various wild type extracts. Sporulation of the wild type can be similarly altered by environmental changes. Although most mutants segregate as single gene mutants in crosses, the mutant progeny phenotypes exhibit various degrees of expression within the cross. Most mutants complement with each other and other morphological mutants of *A. nidulans* to form wild type conidial apparatus, and are recessive to wild type alleles in heterokaryons. A few asporogenous mutants which are invasive appear to be dominant. Twenty loci have been identified so far by complementation. Many mutants are auxotrophic but may be pleiotropic for many functions.

GENETICS OF PROTEASES IN *ASPERGILLUS NIDULANS*

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Mycelium of *Aspergillus* contains a number of electrophoretically distinct proteolytic enzymes; two of these become extracellular during growth on nitrogen-poor media. Mutants deficient in extracellular protease have been isolated and three loci, *xprA*, *xprB* and *xprC* have been defined in linkage groups VIII, VII and VIII. These mutations are recessive in heterokaryon and diploid. Five further complementation groups have been tentatively defined, but exhaustive search for xpr$^-$ mutants has not been made. The mutant *xprC*1, which is stable and easy to classify has been found to involve loss of the two extracellular proteases, both intra- and extracellularly.

The release of extracellular proteases is repressed by low concentrations of nitrogen sources, for example ammonium repression is detectable at 2mM and is complete at 10mM (plate tests). This effect is not due to inhibition of enzyme activity, but it has not yet been shown to be due to reduced enzyme synthesis. Mutants insensitive to repression by nitrate and ammonium are being characterised. One of them (*xprD*1) is of special interest (Cohen, *Journal of General Microbiology* 1972, in press), being completely derepressed for protease under all conditions so far tested. This mutant is simultaneously derepressed for nitrate reductase, xanthine dehydrogenase and glutamate uptake and is semidominant or dominant. It is not methylamine-resistant, nor are methylamine-resistant strains derepressed for protease release. It seems possible that the *xprD* gene plays a key role in general ammonium repression.

CONTROL OF NUCLEIC ACID SYNTHESIS IN *ASPERGILLUS NIDULANS*

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We have continued studying nucleic acid synthesis in *Aspergillus nidulans in vivo* (*Heredity 26*, 346, 1971 and *Biochem. J.*, in press, 1972), using ^{3}H-leucine incorporation as a measure of growth and measuring protein synthesis and nucleic acid synthesis in parallel experiments by following ^{14}C-leucine and ^{14}C-uridine incorporation, respectively. Addition of cycloheximide, anisomycin or emetine, or starvation for a required amino acid, results in parallel decreases in protein and nucleic acid syntheses. In contrast, starvation for inositol drastically reduced nucleic acid synthesis while causing only a marginal reduction in protein synthesis. The significance of these results will be discussed.

POLYMORPHISM AND THE BIOLOGY OF POPULATIONS

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Visible and cytological polymorphisms are a special case among the total range of high-frequency genetical variants at the molecular level. The selection coefficients, of the order of ten per cent., discovered to act upon them, probably give a distorted view of the strength of selection normally acting on alternative alleles. On the other hand, the study of these strongly selected polymorphisms has revealed the wealth of mechanisms by which alleles may be balanced in a population. Unfortunately, we have no idea which balancing mechanisms are the most prevalent, nor a full treatment of their relation with random drift.

Despite the partial failure of Fisher's fundamental theorem and Wright's theorems of increasing $\overline{W}$ (decreasing load) when applied to population ecology, experiments on *Drosophila* populations show a tendency for fitness, as measured by biomass, population size, productivity and homeostasis, to increase under the action of selection.

This effect is associated with increases in heterozygosity, and is probably due to the prevalence of soft selection and, particularly, population dependent selection, rather than to a decrease in the hard genetic load. Heterozygosity also seems to increase short term (physiological) and long term (evolutionary) flexibility. Thus the requirements for immediate adaptedness and for a long term strategy of adaptability may not be in conflict.

EVIDENCE FOR MECHANISMS MAINTAINING POLYMORPHISMS IN HUMAN POPULATIONS

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Evidence for mechanisms maintaining polymorphisms in human populations is sparse and, to some extent at least, controversial. This may well be in part because of the paradox that careful and detailed studies of possible selective mechanisms can only be made in areas which have reached a certain level of economic development and hence availability of medical care; *ipso facto*, types of mortality and morbidity in which polymorphic variation may play a major differential role are relatively rare in such areas. Specific studies of potentially lethal infectious diseases which were until recently one of the main determinants of differential mortality may well prove more rewarding even despite the handicap that they have to be carried out in less privileged areas and must perforce be of simple design and on a relatively small scale.

The evidence which is available is of several kinds, in general associating polymorphic variation with morbidity, fertility, mortality, differential age incidence, and gametic and zygotic selection. Some tentative conclusions may be drawn from this evidence but it should be stressed that these approaches have considerable limitations in that the hypotheses which can be tested probably represent over-simplifications, and, in addition, it is difficult to extrapolate from these findings to the situation in past centuries when the present distribution of polymorphic variations was to a large extent determined.

SMALL SCALE DISRUPTIVE SELECTION IN THE MAINTENANCE OF POLYMORPHISMS

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Large changes in environment can occur over very short distances. There is now plenty of evidence that these can cause highly localised differentiation within species even between populations between which there is considerable gene flow. In plant species with powers of vegetative propagation differentiation can occur between groups of individuals only a few centimetres apart.

This can be a major cause of polymorphism which may pass undetected unless appropriate sampling procedures are adopted. It can lead to excess of heterozygotes.

POLYMORPHISM AND THE AGENTS OF SELECTION

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In the early years of the study of genetical polymorphism it was noticeable how frequently one dominant selective agent (usually biotic) could be identified. Because the genetical models which explain a stable polymorphism require a balance of selective forces it has often been necessary to invoke some counteracting physiological selection in order to accommodate these models. In few cases has the precise nature

of the physiological selection been determined and consequently considerable scepticism has been expressed over the explaining away of apparent difficulties.

With the polymorphism of cyanogenesis in *Trifolium repens* and *Lotus*, however, it is clear that there are interactions between a physiological character and known physical and biotic components of the external environment. Indeed, not only has a selective process of a physiological nature been demonstrated, but the chemistry involved is also well understood.

The role of a third selective agent has recently been described (Foulds and Grime, *Heredity*, *28*, 1972) and although parallel studies in the laboratory and in the field are mutually corroborative, the precise mechanisms have still to be clarified. In consequence wild populations of these plants are being examined for the effect of the three selective agents of temperature (climatic), grazing (biotic) and droughting conditions (edaphic). The hypothesis evolving at the moment regards the response to selection by temperature as strategic, while local tactics are related to animal grazing and soil water conditions.

NICHE EXPLOITATION IN GRASSLAND SYSTEMS

J. L. HARPER

School of Plant Biology, University of North Wales, Bangor

The problem of maintaining balanced polymorphisms within populations and the problem of the mechanisms by which multi-specific diversity is maintained in natural populations have many analogies. These are explored with particular reference to the mechanisms by which population size and niche occupancy are regulated in some grassland communities.

BIOCHEMICAL POLYMORPHISM IN ANIMALS

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No abstract received.

SELECTION VERSUS RANDOM DRIFT IN THE MAINTENANCE OF POLYMORPHISM

B. C. CLARKE

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The lecture reviewed the evidence for and against the large scale involvement of natural selection in the maintenance of polymorphism.