the steel is roughly $10^{-3}$ centimeter. If we assume that the helium is produced uniformly in the alloy and that the precipitate as well as the grain boundary acts as a trap for the gas, then we may calculate the amount of gas that can get to the grain boundary, on the basis of the model chosen for the motion of the gas atoms or bubbles.

The picture I have presented is that of radiation damage, at high temperatures, that involves predominantly the effects of helium produced by $(n,\alpha)$ reactions. This damage appears to be quite general; that is, it occurs in a large number of metals and alloys. However, other mechanisms of radiation damage are known in some alloys in the temperature range from 400° to 650°C. Some workers (*20*) have observed precipitation of compounds in alloys that occurs only in irradiated material. It is possible that, with certain alloys and under certain conditions of irradiation, these precipitates may produce large changes in the properties of the alloy, in addition to the changes due to the helium.


## Summary

The successful development of nuclear power reactors that are economically competitive with other sources of energy has led us to believe that more economical reactors will be developed. But, in developing the next generation of reactors, a new set of problems must be overcome. One of the most important of these is that of the embrittlement of the structural materials at high temperatures as a result of the intense neutron fields in these advanced systems.

The radiation-induced embrittlement at high temperatures is probably associated with helium produced in the materials due to $(n,\alpha)$ reactions with the metal, and in some alloys radiation-induced precipitation of compounds within the alloy may also play a role. We believe that the most serious long-term problem is the generation of helium. Our current understanding of the mechanism by which this radiation damage is produced has allowed us to effect some improvement in the behavior of conventionally produced structural alloys, through minor modifications of the normal working and annealing processes used in their manufacture. However, we may find that new alloys will have to be developed to withstand the service conditions in future nuclear power reactors.


### References and Notes


1. E. P. Wigner, *J. Appl. Phys.* **17**, 857 (1946).
2. For an excellent review of the fundamentals of this type of radiation damage, see F. Seitz, *Science* **138**, 563 (1962).
3. N. E. Hinkle, *Amer. Soc. Testing Mater. Spec. Tech. Pub. No. 341* (1963), p. 344.
4. F. C. Robertshaw, J. Moteff, F. D. Kingsbury, M. A. Pugacz, *ibid.*, p. 372.
5. D. R. Harries and A. C. Roberts, *Nature* **200**, 772 (1963).
6. W. R. Martin, J. R. Weir, R. E. McDonald, J. C. Franklin, *ibid.* **208**, 73 (1965).
7. W. R. Martin and J. R. Weir, paper presented at the 69th annual meeting of the American Society for Testing and Materials, June 1966.
8. C. Zener, *Elasticity and Anelasticity of Metals* (Univ. of Chicago Press, Chicago, 1948), p. 158.
9. J. Moteff, in *Radiation Effects in Materials* (Gordon and Breach, New York, 1966); M. J. Makin, *ibid.*
10. W. R. Martin and J. R. Weir, *Amer. Soc. Testing Mater. Spec. Tech. Pub. No. 380* (1965), p. 251.
11. J. B. Rich, G. P. Walters, R. S. Barnes, *J. Nucl. Mater.* **4**, 287 (1961); C. E. Ells, *ibid.* **5**, 147 (1962); J. R. Weir, in *International Conference on the Metallurgy of Beryllium, London* (Chapman & Hall, London, 1963), pp. 395–409.
12. P. R. B. Higgins and A. C. Roberts, *Nature* **206**, 1249 (1965).
13. D. E. Rimmer and A. H. Cottrell, *Phil. Mag.* **2**, 1345 (1957).
14. A. H. Cottrell, *Met. Rev.* **1**, 479 (1956).
15. P. Vela and B. Russell, *J. Nucl. Mater.* **19**, 312 (1966).
16. D. R. Harries, *J. Brit. Nucl. Energy Soc.* **5**, 74 (1966).
17. H. Alter and C. E. Weber, *J. Nucl. Mater.* **16**, 68 (1965).
18. W. R. Martin and J. R. Weir, *ibid.* **18**. 108 (1966).
19. I. S. Levy and K. R. Wheeler, "Metallographic Evidence of Strain and Irradiation-Induced Precipitation in Preconditioned Hastelloy X-280," *Battelle Northwest Laboratory Rep. No. 211* (1966).
20. D. R. Arkell and P. C. L. Pfeil, *J. Nucl. Mater.* **12**, 145 (1964); E. E. Bloom, W. R. Martin, J. O. Stiegler, J. R. Weir, *ibid.*, in press.


21. I am indebted to J. O. Stiegler for the electron micrographs and to many others of the staff of the Oak Ridge National Laboratory for their assistance in the preparation and review of the manuscript. The research discussed is sponsored by the U.S. Atomic Energy Commission under contract with Union Carbide Corporation.


# Evolutionary Significance of Metabolic Control Systems

## The $\beta$-ketoadipate pathway provides a case history in bacteria.


J. L. Cánovas, L. N. Ornston, R. Y. Stanier


When a metabolic pathway of considerable complexity occurs in several different groups of organisms, biologists are sometimes tempted to assume without further evidence that the pathway in question has had a single evolutionary origin and can thus serve as a marker of evolutionary affinities. A characteristic example of such reasoning can be found in Vogel's review of the biological distribution of the diaminopimelic and $\alpha$-aminoadipic pathways for lysine biosynthesis (*1*). This class of assumptions about evolutionary origin could be rigorously tested by ascertaining whether the enzymes of a pathway which have the same catalytic function in different organisms show extensive homologous amino acid sequences. However, relatively few proteins have been systematically compared in this fashion; cytochrome *c* (*2*) and hemoglobin (*3*) are the most carefully studied examples. For the most part, attempts to distinguish between analogous and homologous enzymes have been less direct and have involved comparisons of molecular weight, amino acid composition, catalytic behavior, or immunological properties (*4*). We believe that the evolutionary significance of the presence of a given biochemical pathway in representatives of several different biological groups can be assessed by means of a somewhat different kind of analysis—comparison of control mechanisms.


Dr. Cánovas is in the department of bacteriology and immunology, University of California, Berkeley, on leave from the Instituto de Biologia Celular, Consejo Superior de Investigaciones Cientificas, Madrid, Spain; Dr. Ornston is in the department of biochemistry, University of Leicester, Leicester, England; Dr. Stanier is a member of the department of bacteriology and immunology, University of California, Berkeley.

## The β-Ketoadipate Pathway and its Control in Pseudomonads

We propose to discuss the β-ketoadipate pathway (Fig. 1) (*5*). Its two convergent branches serve, in many aerobic bacteria, for the oxidative dissimilation of a number of aromatic compounds, during which the six carbon atoms of the benzene nucleus are converted to succinate and acetyl coenzyme A (CoA). So far as is known, the enzymes operative in it have no other functions in the cell. Accordingly, the pathway is a highly specialized one, and the enzymes which catalyze it are inducible in all bacteria so far examined.

In the following discussion, we shall use abbreviations to refer to some of the enzymes of the β-ketoadipate pathway, which bear long and clumsy names. The abbreviations are: carboxymuconate lactonizing enzyme, CMLE; carboxymuconolactone decarboxylase, CMD; enol-lactone hydrolase, ELH; and β-ketoadipate succinyl-CoA transferase, TR.

Ornston (*6*) analyzed the regulation of the synthesis of these enzymes in *Pseudomonas putida* (Fig. 2). Probable inducers were inferred from analysis of the responses of the wild-type organisms to various inducer-metabolites in the pathway and from the modifications of response that occur in mutants which cannot synthesize one or more specific enzymes operative in the pathway. The nature and extent of coordinate regulation of enzyme synthesis was ascertained primarily by systematic comparisons of enzyme activities in cells of the wild type, in which absolute specific activities of the enzymes of the pathway had been varied widely by cultivation under many different conditions of induction and catabolite repression. Coordinate control could be independently deduced from the properties of certain mutants with specific blocks in the pathway.

In *P. putida*, β-ketoadipate (or possibly β-ketoadipyl-CoA), acts as a product-inducer of a coordinate block containing the three enzymes CMLE, CMD, and ELH (*7*). The first two of these enzymes function only in the protocatechuate branch of the β-ketoadipate pathway; but in view of the fact that their synthesis is induced by a metabolite common to both branches, exposure of cells to a metabolic precursor of the catechol branch (for example, benzoate) leads to their full induction. Gratuitous induction of CMLE and CMD as a result of growth on benzoate accordingly provides an indication of the coordinate, product-mediated synthesis of CMLE, CMD, and ELH. Ornston showed that it occurred in two other *Pseudomonas* spp., P. *aeruginosa* and *P. multivorans* (Table 1). Unlike *P. putida*, these two species can use adipate as a substrate for growth; Ornston further found that adipate-grown cells are induced for CMLE, CMD, and ELH. Under these circumstances, the synthesis of all three enzymes is presumably gratuitous; it is no doubt triggered by the formation of either β-ketoadipyl-CoA or β-ketoadipate as an intermediate in adipate dissimilation.

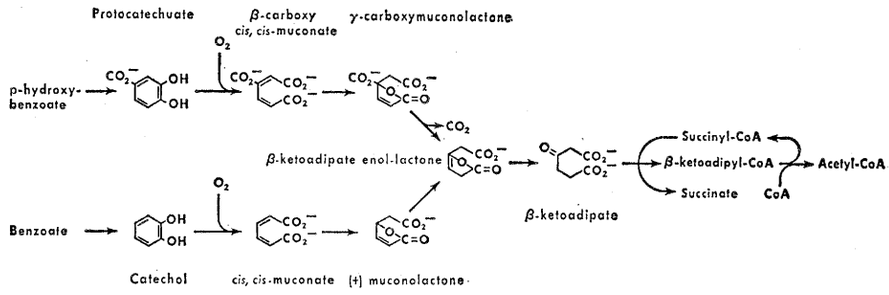


Fig. 1. β-Ketoadipate pathway.

## β-Ketoadipate Pathway and its Control in Other Bacterial Groups

Ornston also examined the patterns of induction of CMLE, CMD, and ELH in *Hydrogenomonas eutropha* and *Moraxella calcoacetica*, two representatives of other genera which can dissimilate benzoate and *p*-hydroxybenzoate through the β-ketoadipate pathway. The first two of these enzymes were not gratuitously induced in either species by growth on benzoate, although this substrate elicited extensive ELH synthesis. Furthermore, growth on adipate failed to elicit significant induction of any of the three enzymes (Table 1). These preliminary observations led us to study in detail the regulation of the enzymes of the β-ketoadipate pathway in *Moraxella calcoacetica.*

Although similar in many nutritional and physiological respects to the members of the *Pseudomonas* group, *M. calcoacetica* differs in structural respects (cell form and absence of flagella) and in the base content of its DNA. The guanosine-cytosine content of DNA from *M. calcoacetica* is approximately 41 moles percent (*7*); that of the DNA from *P. putida, P. aeruginosa*, and *P. multivorans* lies in the range from 62 to

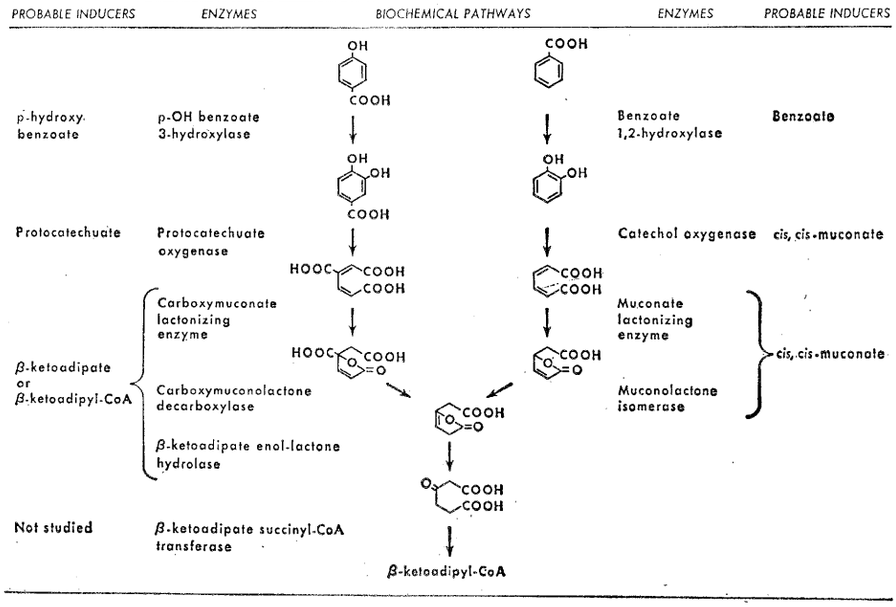


Fig. 2. Regulation of the β-ketoadipate pathway in *Pseudomonas putida,* from the data of Ornston (*6*). Bracketed enzymes are coordinately controlled.

67 moles percent (*8*). This divergence with respect to base content of DNA suggests that the evolutionary separation between *Pseudomonas* and *Moraxella* is a wide one.

We first undertook a systematic analysis of the extent of coordinate regulatory control of the enzymes of the β-ketoadipate pathway in *M. calcoacetica*. In experiments where *p*-hydroxybenzoate or protocatechuate was used as an inducer-substrate, and succinate or acetate as a catabolite repressor, the five enzymes responsible for the conversion of protocatechuate to β-ketoadipyl-CoA invariably showed strict coordination, and none of the enzymes specifically operative in the catechol branch of the pathway was significantly induced. However, when analogous experiments with benzoate or *cis,cis*-muconate as the inducer-substrate were performed, the results likewise indicated strict coordination of the four enzymes responsible for the conversion of *cis,cis*-muconate to β-ketoadipyl-CoA, and none of the enzymes specific for the protocatechuate branch was significantly induced. These seemingly contradictory results could best be explained by assuming that *M. calcoacetica* can synthesize two sets of isofunctional enzymes, mediating common step reactions of the pathway (conversion of β-ketoadipate enol-lactone through β-ketoadipate to β-ketoadipyl-CoA), each set of enzymes being subject to different, coordinate regulatory control. We therefore compared the kinetics of thermal inactivation of the ELH and TR activities in crude extracts of *M. calcoacetica*, prepared from cells grown on benzoate and on *p*-hydroxybenzoate. As a control, parallel experiments were performed with extracts of *P. putida*, prepared from cells grown on the same two substrates. It had previously been shown by several different means (*5*, *9*) that *P. putida* can synthesize only one enzyme with ELH activity. The results of this series of experiments (Table 2) suggest the existence of two sets of isofunctional enzymes under separate control in *M. calcoacetica*; and at the same time they indicate that only one set is synthesized by *P. putida*. We shall designate the enzymes in *p*-hydroxybenzoate-grown cells of *M. calcoacetica* as ELH I and TR I, and those in benzoate-grown cells of this species as ELH II and TR II.

Evidence for the duality of the control of transferase function in *M. calcoacetica* is provided by the properties of two nitrosoguanidine-induced mutants. One has lost the ability to grow with *p*-hydroxybenzoate, but grows normally with benzoate; enzymatic analysis shows that the failure to grow with *p*-hydroxybenzoate is specifically attributable to the loss of TR I. The other has lost the ability to grow with benzoate, but can grow normally with *p*-hydroxybenzoate; enzymatic analysis shows that it is unable to synthesize TR II.

When *M. calcoacetica* is grown with adipate, the activity of β-ketoadipate succinyl-CoA transferase is significantly increased, but is unaccompanied by an increase in the activity of any other enzymes operative in the β-ketoadipate pathway. This activity is attributable to a third enzyme (TR III) that has transferase function. Unlike TR's I and II, TR III is severely and competitively inhibited by adipate; in fact, the $K_I$ for adipate is some two orders of magnitude lower than the $K_M$ for β-ketoadipate. Hence, TR III can be most plausibly interpreted as an adipate-activating enzyme which also has a low affinity for β-ketoadipate; it appears to be induced by adipate or possibly by an early intermediate in adipate dissimilation.

Experiments with *M. calcoacetica* indicate that synthesis of the coordinate block of enzymes in the protocatechuate pathway is induced by protocatechuate, while synthesis of the coordinate block in the catechol pathway is induced by *cis,cis*-muconate. Each coordinate block includes an enzyme which activates β-ketoadipate (TR's I and II, respectively). Transferase function is presumably essential for the dissimilation of β-ketoadipate. However, the control of the synthesis of TR's in *M. calcoacetica* by metabolic precursors of the substrate suggests that β-ketoadipate itself might be incapable of inducing transferase activity and hence incapable of serving as a substrate for the growth of this bacterium. The wild type of *M. calcoacetica* is wholly impermeable to β-ketoadipate and to the other nonaromatic intermediates of the pathway. This barrier can be overcome indirectly by the selection of spontaneous "permeability" mutants, which have acquired the ability to grow on *cis,cis*-muconate. After appropriate induction (for example, by growth on benzoate or *p*-hydroxybenzoate), permeability mutants can oxidize β-ketoadipate at a high rate. Such cells grow linearly with β-ketoadipate, although their growth with *cis,cis*-muconate is exponential. This observation confirms the inability of β-ketoadipate to act as an inducer for *M. calcoacetica*. *Pseudomonas putida*, on the other hand, grows exponentially with β-ketoadipate, even though its TR is likewise under strictly inducible control; accordingly, β-ketoadipate can function both as a substrate and as an inducer (or the metabolic precursor of one) for the β-ketoadipate succinyl-CoA transferase of this species.

Table 1. Influence of the growth substrate on the induction of carboxymuconate lactonizing enzyme (CMLE), carboxymuconolactone decarboxylase (CMD), and enol-lactone hydrolase (ELH) in several species of bacteria. The minus symbol indicates the basal level of enzymatic activity; the plus symbol indicates induced activity at least 20 times the basal level.

| Enzyme activities | Growth substrate: Succinate | *p*-Hydroxybenzoate | Benzoate | Adipate |
|---|---|---|---|---|
| | *P. putida* | | | |
| CMLE, CMD, ELH | − | + | + | |
| | *P. aeruginosa, P. multivorans* | | | |
| CMLE, CMD, ELH | − | + | + | + |
| | *H. eutropha, M. calcoacetica* | | | |
| CMLE, CMD | − | + | − | − |
| ELH | − | + | + | − |

## Constitutive Mutants in Moraxella

In *Moraxella calcoacetica*, β-ketoadipate should serve as a selective substrate for the isolation (from permeability mutants) of mutants that have acquired a constitutive TR function. Normal growth with β-ketoadipate could in principle result from constitutive synthesis of any one of the three TR's; and we have in fact succeeded in isolating mutants constitutive for each of these isofunctional enzymes.

Mutants constitutive for TR I, se-

Table 2. Rates of thermal inactivation of enol-lactone hydrolase (ELH) and β-ketoadipate succinyl-coenzyme A transferase (TR) activities in extracts of *Moraxella calcoacetica* and *Pseudomonas putida*, prepared from cells grown on benzoate and on *p*-hydroxybenzoate.

| Enzymatic activity | Temperature of inactivation (°C) | Half-life of activity (min) in cells grown on: Benzoate | *p*-Hydroxybenzoate |
|---|---|---|---|
| | *M. calcoacetica* | | |
| ELH | 47 | 2 | >30 |
| TR | 50 | 14 | 6 |
| | *P. putida* | | |
| ELH | 49 | 10 | 10 |
| TR | 39 | 12 | 12 |

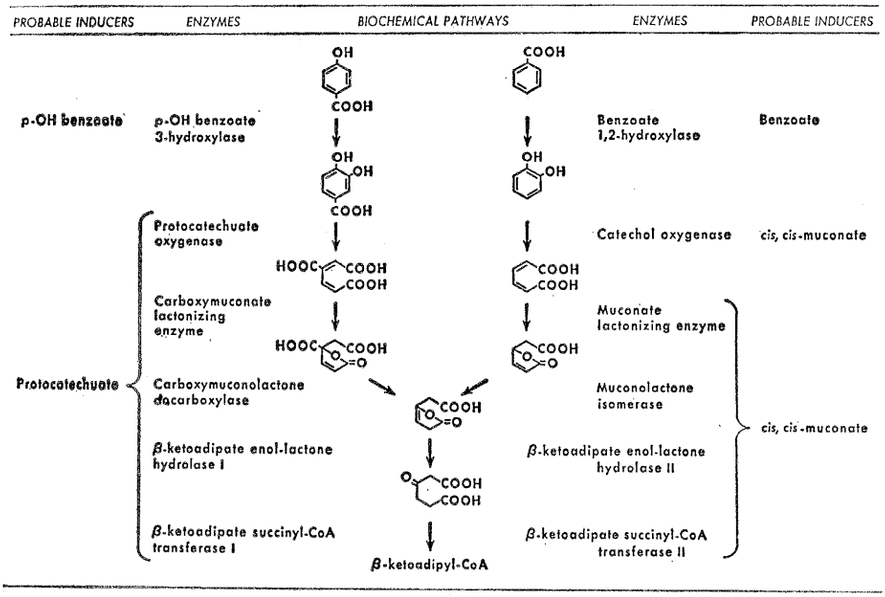


Fig. 3. Regulation of the β-ketoadipate pathway in *Moraxella calcoacetica*. Bracketed enzymes are coordinately controlled.

lected by plating permeability mutants on β-ketoadipate, also synthesize constitutively CMLE, CMD, and ELH I, but without exception have lost protocatechuate oxygenase function. The properties of this class of mutants independently confirm coordination of enzymes of the protocatechuate block. Constitutives of this phenotype have also been isolated indirectly, by selecting mutants unable to synthesize protocatechuate oxygenase from the wild type, after treatment with chemical mutagens. There is an invariable correlation between loss of function for the first enzyme of this coordinate block and constitutivity for the four remaining enzymes of the block. Physiological analyses of many different mutants of this class have shown that their capacity for constitutive enzyme synthesis is not the consequence of a mutation that affects a regulatory gene. It is caused by the endogenous synthesis and accumulation of the inducer, protocatechuate, produced from hydroaromatic intermediates in the pathway of biosynthesis of aromatic amino acids. This accumulation is a direct consequence of the loss of protocatechuate oxygenase function.

Mutants constitutive for TR III can be readily obtained by repeated alternate cultivation of permeability mutants in media containing β-ketoadipate and *p*-hydroxybenzoate as sole carbon sources. This procedure counterselects TR I constitutives, because they cannot grow at the expense of *p*-hydroxybenzoate, as a result of the loss of protocatechuate oxygenase function. The TR III constitutives are still inducible for all enzymes of the β-ketoadipate pathway, which confirms that TR III has no physiological role in this pathway.

Mutants constitutive for TR II have never been obtained directly from permeability mutants by selection for growth on β-ketoadipate. Their isolation required the preliminary construction of a triple mutant, which was permeable to β-ketoadipate and unable to synthesize either TR I or III. When subjected to selection for growth at the expense of β-ketoadipate, this triple mutant yielded mutants constitutive for TR II. These TR II constitutives have also acquired constitutivity for catechol oxygenase, muconate lactonizing enzyme, muconolactone isomerase, and ELH II, and thus provide an independent confirmation of extensive coordination in the catechol branch of the pathway.

Figure 3 summarizes our present picture of the regulatory control of the enzymes of the β-ketoadipate pathway in *M. calcoacetica*. A comparison with the corresponding control map for *P. putida* (Fig. 2) shows a few points of similarity, but many major differences. The most distinctive common feature is the role played by *cis,cis*-muconate as an inducer in the catechol branch of the pathway. In both species, this compound acts as a product-inducer of catechol oxygenase and as a substrate-inducer for a coordinate block of enzymes that includes muconate lactonizing enzyme and muconolactone isomerase. In *P. putida*, the coordinate block comprises only muconate lactonizing enzyme and muconolactone isomerase, both specifically functional in the catechol branch of the pathway; in *M. calcoacetica* it extends to include ELH II and TR II as well.

The differences with respect to control of the protocatechuate branch are much greater. In *P. putida*, protocatechuate can induce synthesis only of protocatechuate oxygenase, the enzyme for which it is the substrate; a sequential induction (in the sense of a shift in the chemical nature of the inducer) immediately follows. In *M. calcoacetica*, protocatechuate is the inducer-substrate for a coordinate block composed of five enzymes: protocatechuate oxygenase, CMLE, CMD, ELH I, and TR I. Its control accordingly extends beyond the point of metabolic convergence. The extended coordination characteristic of *M. calcoacetica* necessitates the synthesis of isofunctional enzymes to mediate step-reactions common to both branches of the pathway. The more limited coordination of control in *P. putida* avoids this particular complication; but the control mechanism operative around the point of metabolic convergence in *P. putida* is likewise a seemingly awkward one, in the sense that it leads to the synthesis of nonfunctional proteins under certain conditions of induction.

## Evolutionary Interpretations

What conclusions can be drawn from this case history? Coordinate product-induction of CMLE, CMD, and ELH seems to be a characteristic feature of control of the β-ketoadipate pathway in the genus *Pseudomonas*, to judge from its occurrence in the three species so far examined. It should be emphasized that these species differ from one another in many unrelated phenotypic respects (*10*), so that their genetic distinctness is unquestionable; furthermore, although *P. aeruginosa* and *P. multivorans* are closely similar in the base content of their DNA, the guanosine-cytosine content of the DNA of *P. putida* is significantly lower (*8*).

The presence of a common control mechanism in three species of the genus *Pseudomonas* acquires much greater significance in view of the fact that the corresponding enzymes of the pathway in *Moraxella calcoacetica* are sub-

ject to an entirely different system of control. These findings suggest that (i) control mechanisms might be relatively stable characters, which tend to be conserved during the evolution of a particular biological group; and (ii) that different control systems governing a given pathway in two biological groups indicate either wide evolutionary divergence or possibly separate evolutionary origins for the pathway in question.

A complex and specialized biochemical pathway might have arisen on several independent occasions in the evolution of a group as ancient as the bacteria. Purely chemical factors severely restrict the play of natural selection in biochemical evolution. For example, if natural selection has to solve the problem of converting the benzene nucleus into aliphatic products that can enter the universal central pathways of cellular metabolism, there are doubtless very few chemically permissible solutions. Accordingly, several different genetic constitutions might each provide favorable evolutionary raw material for achieving the same chemical solution of this physiological problem. If such were the case, the evolutionary end products would appear homologous on the metabolic level, even though the operative enzymes and the corresponding structural genes were nonhomologous.

The development of the relevant set of structural genes is a necessary, but not a sufficient, condition for the evolution of a new biochemical pathway. Physiological integration of the enzymes must follow, and it requires the imposition of a novel pattern of control, with the metabolites of the pathway as effectors. However, not every metabolite needs to be endowed with effector function in order to maintain a well-regulated complement of enzymes (Figs. 2 and 3). A considerable element of choice must occur at this stage in the evolution of a pathway, both with respect to the metabolites which actually acquire effector function, and with respect to the precise manner in which they exercise it. One can readily imagine that the genetic background could influence strongly the choice which is made. Hence, independent evolution of a biochemical pathway in two groups is apt to be mirrored in differences at the level of control systems.


**References**

1. H. J. Vogel, in *Evolving Genes and Proteins*, V. Bryson and H. J. Vogel, Eds. (Academic Press, New York, 1965), p. 25.
2. E. Margoliash and E. L. Smith, *ibid.*, p. 221.
3. J. Buettner-Janusch and R. L. Hill, *ibid.*, p. 167.
4. N. O. Kaplan, *ibid.*, p. 243.
5. L. N. Ornston and R. Y. Stanier, *J. Biol. Chem.* **241**, 3776 (1966).
6. L. N. Ornston, *ibid.*, p. 3800.
7. M. Mandel, personal communication.
8. ———, *J. Gen. Microbiol.* **43**, 273 (1966).
9. L. N. Ornston, *J. Biol. Chem.* **241**, 3787 (1966).
10. R. Y. Stanier, N. Palleroni, M. Doudoroff, *J. Gen. Microbiol.* **43**, 159 (1966).


11. Supported by grant AI-1808 from the National Institute of Allergy and Infectious diseases, PHS.


# The Pure-Science Ideal and Democratic Culture

## A new scientific ideal in the late 19th century led to continuing conflicts with democratic assumptions.


George H. Daniels


One of the most notable developments within the scientific community in post-Civil War America was a changed image of the scientist and of his role in society. Previously, science had been "sold" to the public in terms of its contributions to important American values—utilitarian, equalitarian, religious—or even as a means of social control, depending upon the speaker's best estimate of his audience. But in the 1870's, for the first time, great numbers of scientific spokesmen began to vocally resent this dependence upon values extraneous to science. The decade, in a word, witnessed the development, as a generally shared ideology, of the notion of science for science's sake. Science was no longer to be pursued as a means of solving some material problem or of illustrating some Biblical text; it was to be pursued simply because the truth—which was what science was thought to be uniquely about —was lovely in itself, and because it was praiseworthy to add what one could to the always developing cathedral of knowledge.


The author is assistant professor of history at Northwestern University, Evanston, Illinois.


Historians of art, business, the law, or a great many other subjects will readily recognize this ideal, for in one sense it was the scientific analog of the general fragmentation of life and thought that was occurring in the 1870's. That decade also witnessed, to name only two well-known examples, the origin of "art for art's sake," and of "profit for profit's sake," a notion admirably analyzed by Thomas Mann in his novel *Buddenbrooks*.

In science, fugitive expressions of the sentiment can be found from time to time in the literature of the early 19th century, and it is true that practical values cannot be deduced from the nature of the work American scientists of the first half of the century chose to do. Their labors in science, in terms of fields of interest, were probably indistinguishable from those of the postwar generation (*1*). Yet, the claims of practicality by that earlier generation were no mere rhetoric. Those researches that were not immediately practical no doubt soon would be. Even if they were not useful in a material sense, they would serve a valuable public function by further demonstrating the character and power of God. Scientific education, the earlier generation argued, was necessary because "it would powerfully conduce to benefit the morals"; the government