First publ. in: Taurine 6: [proceedings of the 15th International Taurine Meeting, "Taurine Today", held in Tampere, Finland, June 12 - 15, 2005 / ed. by Simo S. Oja and ... New York: Springer, 2006, pp. 3-13

METABOLISM OF TAURINE IN MICROORGANISMS

A Primer in Molecular Biodiversity?

Alasdair M. Cook and Karin Denger*

1. INTRODUCTION

To those studying the roles of taurine in mammals, one starting point might be Huxtable's review (Huxtable, 1992), in which about one page was devoted to a list of known and putative functions of the compound. Subsequent taurine meetings have added much to this picture. The review also supplies information for microbiologists; in the meantime, much has been learned about the roles of taurine in microbial metabolism, and I see it as my brief to introduce the facts of the matter, and to delineate fact and hypothesis.

First of all, I would like to remind you that we are, from the point of view of weight, mainly eukaryotic. However, from the point of view of numbers, we are largely prokaryotic. To be here, we should be able to perform considerable academic feats involving e.g. nerve cells and taurine, but metabolically, we are dunces: we can excrete it or we can conjugate it and excrete it. It is prokaryotes, apparently the bacteria, which show brilliance in manipulating taurine. So be warned that we are about to jump from one biogeochemical cycle to the next. Please fasten your seatbelts!

One of the first microbiologists to mention taurine was den Dooren de Jong in 1926 (den Dooren de Jong, 1926); he seems to have tested the compound as a source of nitrogen for growth, with success: we will return to the nitrogen cycle later. A key player was Kondo, who, with Shimamoto and Berk, set the scene for much of the present work in the carbon cycle by discovering sulfoacetaldehyde as a key intermediate (cited in Cook and Denger, 2002). Another key player is Kertesz (2000), who established the oxygenolytic desulfonation of taurine in the sulfur cycle. Mammals are used to aerobic conditions, but about half the biosphere (by weight) is anoxic, and many taurine utilizers grow under strictly anoxic conditions (Lie *et al*., 1998; Cook and Denger, 2002): be prepared for novel respirations and fermentations!

* Department of Biological Sciences, The University, D 78457 Konstanz, Germany.

We are used to taurine being the major organic solute in mammals (Huxtable, 1992) but think of the consequences for bacteria. When I drive you to tears, you are excreting taurine to feed bacteria. When I drive you to the toilet, your fluid and solid contributions to the sewage plant contain taurine and conjugated taurine. When I drive you to appendicitis, the taurine could well be utilized by *Bilophila wadsworthia* from your gut canal. When you try to wash me out of your hair, you are using taurine derivatives to generate the mild foam. When you get caught in a spider's web, one glue-auxiliary is taurine. And if you throw yourself into the ocean to escape, you will find taurine as an osmolyte in deep-sea creatures. At least one antibiotic, bulgecin A, is a taurine conjugate. Bacteria have many potential sources of taurine, but presumably usually at low concentrations.

2. TAURINE TRANSPORT INTO THE BACTERIAL CELL

Sulfonation has been described as Nature's way of keeping a compound on one side of a biological membrane (Graham *et al.*, 2002): the permanent negative charge in the physiological pH-range prevents passive diffusion across biological membranes. I gather that mammals have a relatively simple transporter, TauT [TC 2.A.22.3.3], to move taurine across membranes. Characterized enzymes of taurine biotransformation in bacteria are usually soluble, intracellular enzymes, so transport into the cell is essential for bacteria to be able to utilize the compound.

Bacteria seem to have complex transporters. The best-understood transport system for taurine is associated with the assimilation of taurine sulfur in *Escherichia coli*. This is a 3-component ATP-binding cassette transporter, i.e. an ABC transporter termed TauABC [TC 3.A.1.17.1] (Eichhorn *et al.*, 2000). The authors used mutation analysis and complementation studies to confirm the function of all three components. TauA is a periplasmic binding protein, TauC is the permease and TauB is the ATPase. This system, certainly as homologous genes and occasionally with further experimental support, is widespread in bacteria which utilize taurine sulfur for growth (Kertesz, 2001; Masepohl *et al.*, 2001).

We suspect that ABC transporters are also involved in the utilization of taurine as a carbon source. Currently the evidence is purely that of genes homologous to *tauABC* in e.g. *Sinorhizobium meliloti* and *Paracoccus pantotrophus*, and clustered with other genes known or believed to be involved with taurine dissimilation (Brüggemann *et al.*, 2004).

A different multi-component system is also believed to be involved in taurine transport in e.g. *Rhodobacter sphaeroides* and *Paracoccus denitrificans*. This is a tripartite ATP-independent (TRAP) system [TC 2.A.56.4.1] whose genes, *tauKLM*, are clustered with other genes known or believed to be involved with taurine dissimilation (Brüggemann *et al.*, 2004). There is, as yet, no experimental evidence for function, simply a sequence similarity to other TRAP transporters.

3. TWO ROUTES TO GENERATE SULFOACETALDEHYDE

Taurine dehydrogenase (TDH) was discovered by Kondo's group (Kondo *et al.*, 1971) and it is still referred to officially as EC 1.4.99.2, which indicates how difficult the enzyme is to study. Brüggemann et al. (2004) have now established that the physiological

electron acceptor is cytochrome *c*, so we presume that the enzyme may be reclassified as EC 1.4.2.- (Fig. 1). The membrane-bound enzyme has not been purified, and we have not yet confirmed our hypothesis that the *tauXY*-genes encode the structural proteins (Ruff *et al.*, 2003; Brüggemann *et al.*, 2004). Work is in progress to express the genes heterologously. In the meantime, we were forced to postulate that some taurine dehydrogenases (e.g. TauXY in *Rhodopseudomonas palustris*) require a native cytochrome *c* for activity, rather than bovine cytochrome *c* (Denger *et al.*, 2004b). Weinitschke has confirmed this idea by adding cytochrome *c*, which she isolated from *R. palustris*, to crude extract of *R. palustris*, and obtaining deamination of taurine (manuscript in preparation).

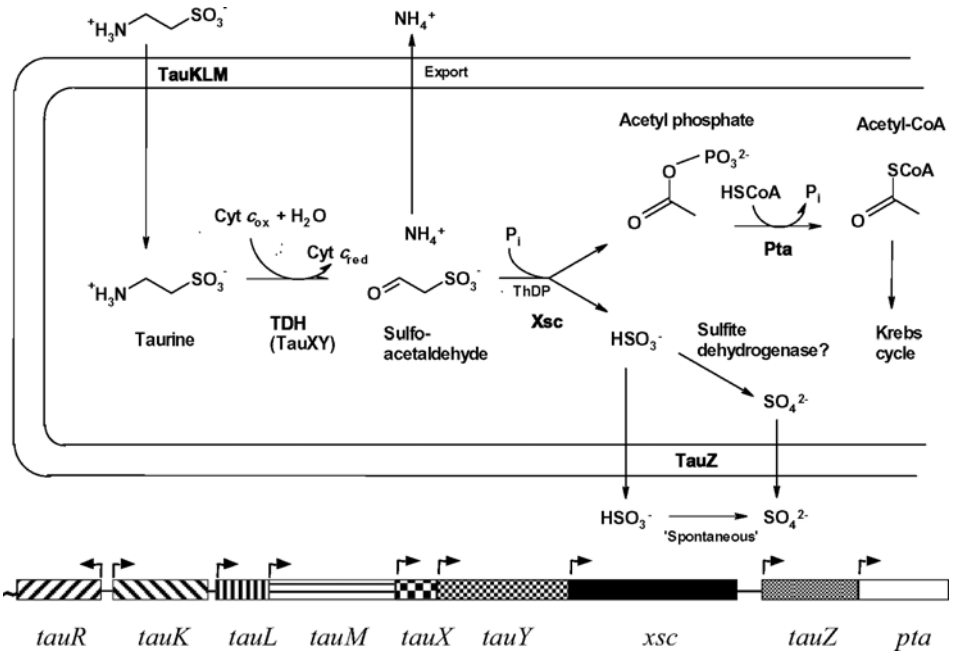

Figure 1. Presumed pathway for the dissimilation of taurine in *Paracoccus denitrificans* NKNIS, and the corresponding genes. Inducible TDH (taurine dehydrogenase), Xsc (sulfoacetaldehyde acetyltransferase) and Pta (phosphotransacetylase) have been assayed and *tauZ* is transcribed inducibly. The abbreviation ThDP represents thiamine diphosphate. The fate of acetyl CoA is shown as the Krebs cycle, but this masks the requirement for anaplerotic enzymes, which in the β Proteobacteria would be the glyoxylate shunt. The nature of the anaplerotic pathway in many α Proteobacteria (e.g. strain NKNIS) is still unknown.

An alternative route to sulfoacetaldehyde is taurine:pyruvate transaminase (Tpa) [EC 2.6.1.77] coupled to alanine dehydrogenase (Ald) [EC 1.4.1.1] (Shimamoto and Berk, 1980). The enzymes were first purified from *Bilophila wadsworthia* and sequenced (Laue and Cook, 2000a,b). More recently, the enzymes were purified from *Rhodococcus* spp. and sequenced (Denger *et al.*, 2004a), but the organism in which the most complete pathway can be sketched is apparently *Silicibacter pomeroyi,* where it is derived from the

genome sequence (Moran *et al.*, 2004) (Fig. 2). Whereas there is a high degree of sequence homology amongst the TauXY sequences (Brüggemann *et al.*, 2004), there is considerable diversity amongst the Tpa sequences (not shown).

One organism, at least *Rhodobacter sphaeroides*, seems to express both the TDH and the Tpa, which can be deduced from the genome sequence (Novak *et al.*, 2004; Denger, 2005). Both reactions have been detected in *Paracoccus pantotrophus* as well (Mikosch *et al.*, 1999; Brüggemann *et al.*, 2004), but this has not yet been explored in detail.

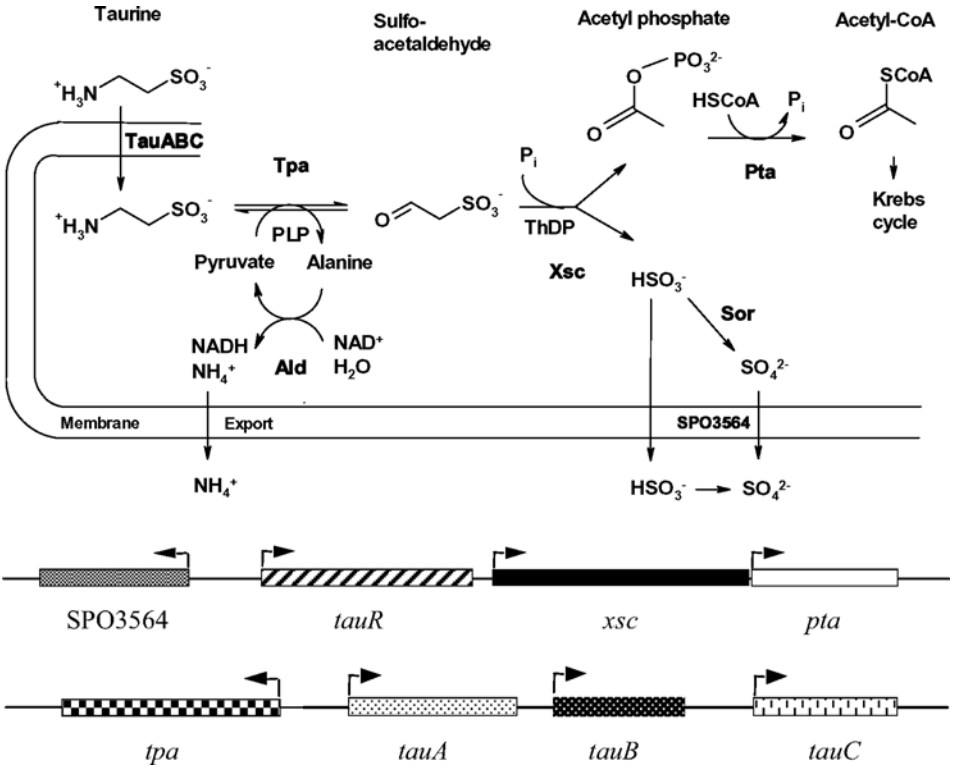

Figure 2. The presumed degradative pathway for taurine in *Silicibacter pomeroyi*^{T}, and the corresponding genes. This pathway is an example with an ABC transporter and a Tpa (taurine:pyruvate aminotransferase). Growth with taurine is quantitative (Denger, 2005). Inducible Ald (alanine dehydrogenase), Xsc (sulfoacetaldehyde acetyltransferase) and Sor (sulfite dehydrogenase) have been measured; neither Sor nor the *sor* gene has been identified (Denger, 2005). The identity of the sulfate exporter (SPO3564) is hypothetical. The abbreviation ThDP represents thiamine diphosphate. The enzymes and transporters appear to be encoded by SPO0673 0676 (*tpa* to *tauC*), SPO3560 3562 (*pta* to *tauR*) and SPO0222 (*ald*).

4. SULFOACETALDEHYDE ACETYLTRANSFERASE

The nature of the desulfonation reaction was identified only recently and shown to be representative for enzymes studied previously (Ruff *et al.*, 2003). Sulfoacetaldehyde acetyltransferase (Xsc) [EC 2.3.3.15] is one of a newly recognized group of acetyltransferases in which the acetyl group is subject to isomerization during transfer.

Otherwise it seems to be a fairly standard thiamine diphosphate-coupled enzyme, which has the great advantage, especially for anaerobes, of yielding a high-energy bond, acetyl phosphate (Figs. 1 and 2). The sulfonate group is released as sulfite.

Several lines of evidence led us to hypothesize, and then to verify, this reaction. In part we discovered the phosphate-dependence of the reaction and in part the sequencing project yielded the neighbouring phosphotransacetylase gene (e.g. Figs. 1 and 2). Literature data pointed out the lability of acetyl phosphate under all sample work-up regimes used previously, so minor alterations in sampling brought dramatic changes in reactants, products and stoichiometry. The new reaction was established, a mechanism suggested (Cook and Denger, 2002) and the older version withdrawn by the Nomenclature Committee (NC-IUBMB).

The enzyme has been found in α-, β-, γ- and δ-Proteobacteria as well as in high- and low-G+C-content Gram-positive bacteria. Up till now we have recognized three subgroups of the enzyme, each of which has been purified and sequenced, and we suspect the presence of either another subgroup or an alternative enzyme type which is unstable (in e.g. *Bilophila wadsworthia*) (Ruff *et al.*, 2003; Brüggemann *et al.*, 2004).

5. PHOSPHOTRANSACETYLASE

The phosphotransacetylase (Pta) (phosphate acetyltransferase [EC 2.3.1.8]), which was first suggested from sequence data (see Chapter 4), could be detected as enzyme activity (Figs. 1 and 2) (Ruff *et al.*, 2003). As yet, direct confirmation has not been provided that the assayed enzyme is the product of the gene sequence indicated in Figs. 1 and 2. Preliminary analyses of sequenced genomes indicate that there are at least two classes of Pta. Indeed, in rare cases we have been unable to assay a phosphotransacetylase. The current hypothesis for the latter observation is that we have the wrong assay conditions, or that a class of Pta is unstable. This problem still needs to be addressed.

6. THE FATE OF TAURINE CARBON

Chapters 2-5 have given examples of aerobic dissimilation of taurine (e.g. the strict aerobe *Silicibacter pomeroyi* in Fig. 2). This is characterized by respiring about 50 % of the taurine to CO_2 and converting the other 50 % to biopolymers in cells. A similar situation holds true in Fig. 1, where the same dissimilative enzymes function whether *Paracoccus denitrificans* NKNIS is respiring with O_2 or with NO_3^- as the terminal electron acceptor. Under these conditions, the acetyl CoA will be processed via the Krebs cycle and an anaplerotic pathway. The latter is presumably the glyoxylate bypass in β-Proteobacteria, where the necessary genes are present in organisms with a sequenced genome (e.g. *Burkholderia xenovorans* LB400), or have been detected by direct assay (Denger and Cook, 2001). The nature of the anaplerotic pathway in several α-Proteobacteria is still unclear (Novak *et al.*, 2004).

A range of strictly anaerobic bacteria dissimilates taurine. There is a sulfite respiration in *Bilophila wadsworthia* and in several sulfate-reducing bacteria (Laue *et al.*, 1997; Lie *et al.*, 1998). There is one fermentation in *Desulfonispora thiosulfatigenes*, another in *Desulforhopalus singaporensis* (Denger *et al.*, 1999; Lie *et al.*, 1999). In all

cases, the major fate of the taurine carbon is acetate; some organisms utilize the remaining carbon for biosynthesis of biopolymers.

Where data are available, it would appear that taurine transaminase is used in these strict anaerobes to generate sulfoacetaldehyde. In very few cases has the presence of Xsc been confirmed (see Chapter 4). The enzyme that can be measured routinely in these organisms is Pta, but in this case it is accompanied by acetate kinase (Ack [EC 2.7.2.1]). We interpret this as conservation of energy by substrate level phosphorylation by Ack to yield ATP and the acetate, which is excreted by an unknown mechanism. Pta converts a portion of the acetyl phosphate generated by the often-putative Xsc to acetyl CoA for biosynthetic purposes.

7. THE FATE OF TAURINE NITROGEN DURING CARBON LIMITATION

When bacteria dissimilate taurine carbon, the ammonium ion is released (Figs. 1 and 2). This ammonium ion is in excess of requirements: bacteria require some 10 mol carbon per mol nitrogen. During growth, some 80% of the ammonium ion is recovered in the growth medium, usually concomitantly with growth, while the remainder is found in cell material (Denger *et al.*, 1997). The nature of the exporter is unknown, though an Amt protein (Khademi *et al.*, 2004) might be appropriate.

8. THE MANY FATES OF TAURINE SULFUR

The initial fate of taurine sulfur is always sulfite (Figs. 1 and 2), whether the organism is strictly anaerobic, facultatively anaerobic or strictly aerobic. The requirement of the cell for sulfur for biosynthetic purposes is negligible (about 1% of cell dry weight), so effectively all the sulfite remains to be processed.

The strictly anaerobic bacteria, which dissimilate taurine, apparently do so to obtain sulfite. These organisms then carry out a sulfite respiration via sulfite reductase (Laue *et al.*, 2001), and many of them excrete the sulfonate moiety as sulfide, although dismutation to sulfide and sulfate is known, as is the release of thiosulfate (Cook and Denger, 2002). In some ways, taurine can be considered as a non-toxic source of sulfite for these organisms (Laue *et al.*, 2001).

Sulfite can be regarded as a dual problem for facultative anaerobes and aerobes. In part there is the aspect of toxicity, and in part there is the problem of the osmotic pressure within the cell and the requirement to maintain constant conditions in the cell.

The problem of toxicity seems to be solved largely by oxidizing the sulfite to sulfate (Cook and Denger, 2002). This simple answer masks a range of problems. Is sulfite dehydrogenase [EC 1.8.2.1] or sulfite oxidase [EC 1.8.3.1] involved? There appears to be no report of sulfite oxidase [EC 1.8.3.1] in bacteria. And how many different sulfite dehydrogenases are there? The characterized bacterial sulfite dehydrogenase (SorAB) is periplasmic (Kappler *et al.*, 2000), which seems unsuitable to dispose of intracellular sulfite, and *sorAB*-like genes of high identity are not widespread in sequenced bacterial genomes. At least one other type of sulfite dehydrogenase is known (Reichenbecher *et al.*, 1999), and we believe this type to be present in *Silicibacter pomeroyi* (Denger, 2005), which does not contain the *sorAB*-genes. A lot of questions remain to be answered here.

The problem of homeostasis seems to be answered by sulfate exporters. We postulate that TauZ (Fig. 1) is a sulfate exporter (Rein *et al.*, 2005), one of many, but proof is still needed. We hypothesize that SPO3564 (Fig. 2) is another sulfate exporter, again without experimental evidence.

9. TAURINE AS A SOLE SOURCE OF NITROGEN FOR BACTERIA

It was clear from earlier work, e.g. Figs. 1 and 2, that taurine-nitrogen was used for growth. But what happens, should one supply a further source of carbon and allow the organism to utilize all the available nitrogen? We did this experiment with *Rhodococcus opacus*, and found that the complete dissimilative pathway for taurine was present and active (effectively as in Fig. 2), but that specific activity of the enzymes was reduced, presumably reflecting the lower requirement for nitrogen compared with carbon. No ammonium ion was released into the medium, so presumably the exporter was inactive (Denger *et al.*, 2004a).

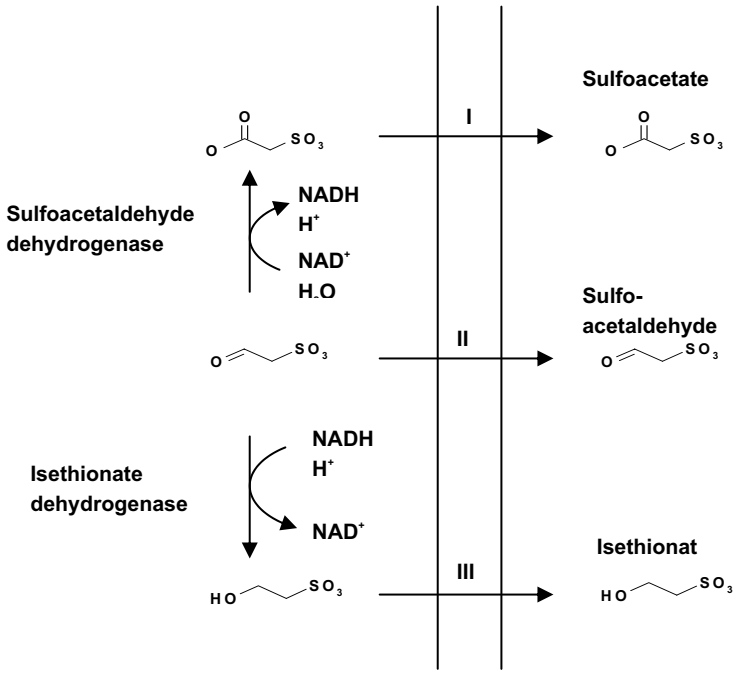

Figure 3. The major fates of carbon and sulfur when taurine is utilized as a sole source of nitrogen by different bacteria. Another fate is sulfate and CO_2, but that was minor in the experiments (Weinitschke *et al.*, 2005). *Rhodopseudomonas palustris* uses taurine dehydrogenase to generate sulfoacetaldehyde, sulfoacetaldehyde dehydrogenase to generate sulfoacetate and we hypothesize exporter I (Denger *et al.*, 2004b). *Acinetobacter calcoaceticus* also uses taurine dehydrogenase to generate sulfoacetaldehyde, which is excreted quantitatively by putative exporter II (Weinitschke *et al.*, 2005). *Klebsiella oxytoca* transaminates taurine to generate sulfoacetaldehyde, which is reduced by isethionate dehydrogenase, and the isethionate excreted by putative exporter III (Styp von Rekowski *et al.*, 2005).

Sequence data led us to test whether *Rhodopseudomonas palstris* utilized taurine nitrogen. It did so, but there was no excretion of sulfate, in contrast to the metabolism of *Rhodococcus opacus*. Instead, the organism excreted sulfoacetate quantitatively (Fig. 3). We thus had a new pathway to generate sulfoacetate, which was previously known only from the degradation of the plant sulfolipid (Denger *et al.*, 2004b).

Further exploration of this phenomenon showed that the release of an organosulfonate from taurine under these conditions was normal; only about 10% of isolates released sulfate (Weinitschke *et al.*, 2005). One of the products formed was sulfoacetaldehyde: as the compound is utilized as a growth substrate by other bacteria (Lie *et al.*, 1996), we presume that the excretion of sulfoacetaldehyde is not unusual.

The third organosulfonate that we discovered in quantitative amounts was isethionate (Styp von Rekowski *et al.*, 2005). The generation of isethionate from taurine in faecal material was known (Fellman *et al.*, 1980), and those authors attribute mammalian isethionate to bacterial production in the gut. We now supply a physiological and biochemical background to that observation.

Just as the excretion of sulfate requires an exporter, in our hypotheses (Chapter 8), we see a requirement for exporters of sulfoacetate, sulfoacetaldehyde and isethionate (Fig. 3). In the latter cases, however, there is even less experimental evidence that in Chapter 8.

10. REGULATION OF INDUCTION

The utilization of taurine described in Chapters 2-9 involves regulation of enzyme induction. In almost all cases where we have data, a gene neighbouring a region encoding a recognized portion of the pathways in Figs. 1-3 is found in common. We have termed it the *tauR*-gene (Figs. 1 and 2), because of its similarity to known transcriptional regulators (Ruff *et al.*, 2003; Brüggemann *et al.*, 2004), but here again, we have not yet tested the hypothesis. The genome of *Desulfotalea psychrophila* (Rabus *et al.*, 2004), with candidates for *tauKLM*, *tpa*, ald and *xsc* (see Figs. 1 and 2), contains no *tauR*-like gene, so a different regulatory protein or mechanism seems likely.

The available evidence indicates that sulfite dehydrogenase is inducible. When an organism utilizes more than one inducible desulfonation pathway (e.g. *Paracoccus pantotrophus* NKNCYSA), the sulfite dehydrogenase is induced in both cases (Rein *et al.*, 2005), which shows that the regulation of sulfite dehydrogenase is independent of the regulation of the degradation of sulfonates.

11. SCAVENGING FOR SULFUR UNDER GLOBAL REGULATION

The requirement for sulfur for biomolecules is orders of magnitude lower than for e.g. carbon (Chapter 8). Correspondingly, different enzymes are needed under the different conditions of sulfur limitation and carbon limitation. Similarly, different regulation is required. Kertesz (2000) describes this in detail.

The regulation is not the specific induction presumed in Chapter 10. Instead, there is global regulation, whereby the cell under sulfate starvation simultaneously switches on all scavenging systems it contains; sometimes, additional regulatory circuits are involved. One of those is for taurine.

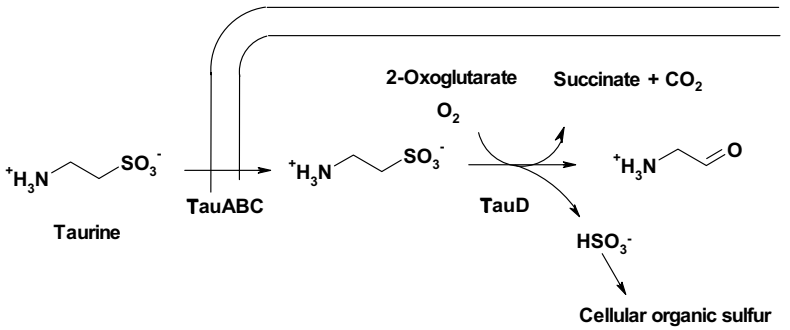

Figure 4. Desulfonation of taurine involving the products of the taurine cluster *tauABCD*, which was initially discovered in *Escherichia coli* (Eichhorn *et al.*, 1997, 2000).

The taurine cluster contains four genes, *tauABCD*. The transporter, TauABC (Fig. 4), was introduced earlier (Chapter 2). The desulfonation is oxygenolytic (Fig. 4). Taurine dioxygenase (TauD [EC 1.14.11.17]) is a 2-oxoglutarate-dependent oxygenase, which generates sulfite, succinate and 2-aminoacetaldehyde. This system is very widespread in bacteria, as a search of the NCBI database with the BLAST algorithm and any of the protein sequences shows.

12. FINAL COMMENTS

The degradative path for taurine is short, but it includes novel biochemistry and multiple transport and regulatory phenomena of general relevance, which are poorly understood and which we hope to elucidate.

We are grateful to John Quinn (Queen's University Belfast, Northern Ireland, UK) for sharing his understanding of sulfonate metabolism with us. The research was funded by the Deutsche Forschungsgemeinschaft, the University of Konstanz, the European Union (SUITE) and the LBS Stiftung 'Umwelt und Wohnen'.

13. REFERENCES

Brüggemann, C., Denger, K., Cook, A. M., and Ruff, J., 2004, Enzymes and genes of taurine and isethionate dissimilation in *Paracoccus denitrificans*, *Microbiology (Reading)* **150**:805 816.

Cook, A. M., and Denger, K., 2002, Dissimilation of the C_2 sulfonates, *Arch. Microbiol.* **179**:1 6.

den Dooren de Jong, L. E. 1926, *Bijdrage tot de kennis van het mineralisatieproces*, Rotterdam: Nijgh and van Ditmar.

Denger, K., 2005, Unpublished data.

Denger, K., and Cook, A. M. 2001, Ethanedisulfonate is degraded via sulfoacetaldehyde in *Ralstonia* sp. strain EDS1, *Arch. Microbiol.* **176**:89 95.

Denger, K., Laue, H., and Cook, A. M., 1997, Anaerobic taurine oxidation: a novel reaction by a nitrate reducing *Alcaligenes* sp, *Microbiology (Reading)* **143**:1919 1924.

Denger, K., Ruff, J., Schleheck, D., and Cook, A. M., 2004, *Rhodococcus opacus* expresses the *xsc* gene to utilize taurine as a carbon source or as a nitrogen source but not as a sulfur source, *Microbiology (Reading)* **150**:1859 1867.

Denger, K., Stackebrandt, E., and Cook, A. M., 1999, *Desulfonispora thiosulfatigenes* gen. nov., sp. nov., a widespread, taurine fermenting, thiosulfate producing, anaerobic bacterium, *Int. J. Syst. Bacteriol.* **49**:1599 1603.

Denger, K., Weinitschke, S., Hollemeyer, K., and Cook, A. M. 2004, Sulfoacetate generated by *Rhodopseudomonas palustris* from taurine, *Arch. Microbiol.* **182**:254 258.

Eichhorn, E., van der Ploeg, J. R., Kertesz, M. A., and Leisinger, T., 1997, Characterization of α ketoglutarate dependent taurine dioxygenase from *Escherichia col, J. Biol. Chem.* **272**:23031 23036.

Eichhorn, E., van der Ploeg, J. R., and Leisinger, T., 2000, Deletion analysis of the *Escherichia coli* taurine and alkanesulfonate transport systems, *J. Bacteriol.* **182:**2687 2795.

Fellman, J. H., Roth, E. S., Avedovech, N. A., and McCarthy, K. D., 1980, The metabolism of taurine to isethionate, *Arch. Biochem. Biophys.* **204**:560 567.

Graham, D. E., Xu, H., and White, R. H., 2002, Identification of coenzyme M biosynthetic phosphosulfolactate synthase: a new family of sulfonate biosynthesizing enzymes, *J. Biol. Chem.* **277**:13421 13429.

Huxtable, R. J. 1992, Physiological actions of taurine, *Physiol. Rev.* **72**:101 163.

Kappler, U., Bennett, B., Rethmeier, J., Schwarz, G., Deutzmann, R., McEwan, A. G., and Dahl, C., 2000, Sulfite:cytochrome *c* oxidoreductase from *Thiobacillus novellus*. Purification, characterization, and molecular biology of a heterodimeric member of the sulfite oxidase family, *J. Biol. Chem.* **275**:13202 13212.

Kertesz, M. A. 2000, Riding the sulfur cycle metabolism of sulfonates and sulfate esters in Gram negative bacteria, *FEMS Microbiol. Rev.* **24**:135 175.

Kertesz, M. A. 2001, Bacterial transporters for sulfate and organosulfur compounds, *Res. Microbiol.* **152**:279 290.

Khademi, S., O'Connell, J., 3rd, Remis, J., Robles Colmenares, Y., Miercke, L. J., and Stroud, R. M., 2004, Mechanism of ammonia transport by Amt/MEP/Rh: structure of AmtB at 1.35 A, *Science (Washington, DC)* **305**:1587 1594.

Kondo, H., Anada, H., Ohsawa, K., and Ishimoto, M.,1971, Formation of sulfoacetaldehyde from taurine in bacterial extracts, *J. Biochem.* **69**:621 623.

Laue, H. and Cook, A. M., 2000, Biochemical and molecular characterization of taurine:pyruvate aminotransferase from the anaerobe *Bilophila wadsworthia*, *Eur. J. Biochem.* **267**:6841 6848.

Laue, H. and Cook, A. M., 2000, Purification, properties and primary structure of alanine dehydrogenase involved in taurine metabolism in the anaerobe *Bilophila wadsworthia*, *Arch. Microbiol.* **174**:162 167.

Laue, H., Denger, K., and Cook, A. M., 1997, Taurine reduction in anaerobic respiration of *Bilophila wadsworthia* RZATAU, *Appl. Environ. Microbiol.* **63**:2016 2021.

Laue, H., Friedrich, M., Ruff, J., and Cook, A. M., 2001, Dissimilatory sulfite reductase (desulfoviridin) of the taurine degrading, non sulfate reducing bacterium *Bilophila wadsworthia* RZATAU contains a fused DsrB DsrD subunit, *J. Bacteriol.* **183**:1727 1733.

Lie, T. J., Clawson, M. L., Godchaux, W., and Leadbetter, E. R., 1999, Sulfidogenesis from 2 aminoethanesulfonate (taurine) fermentation by a morphologically unusual sulfate reducing bacterium, *Desulforhopalus singaporensis* sp. nov, *Appl. Environ. Microbiol.* **65**:3328 3334.

Lie, T. J., Pitta, T., Leadbetter, E. R., Godchaux III, W., and Leadbetter, J. R., 1996, Sulfonates: novel electron acceptors in anaerobic respiration, *Arch. Microbiol.* **166**:204 210.

Lie, T. L., Leadbetter, J. R., and Leadbetter, E. R., 1998, Metabolism of sulfonic acids and other organosulfur compounds by sulfate reducing bacteria, *Geomicrobiol. Rev.* **15**:135 149.

Masepohl, B., Führer, F., and Klipp, W., 2001, Genetic analysis of a *Rhodobacter capsulatus* gene region involved in utilization of taurine as a sulfur source, *FEMS Microbiol. Lett.* **205**:105 111.

Mikosch, C., Denger, K., Schäfer, E. M., and Cook, A. M., 1999, Anaerobic oxidations of cysteate: degradation via a cysteate:2 oxoglutarate aminotransferase in *Paracoccus pantotrophus*, *Microbiology (Reading)* **145**:1153 1160.

Moran, M. A., Buchan, A., González, J. M., Heidelberg, J. F., Whitman, W. B., Klene, R. P., Henriksen, J. R., King, G. M., Belas, R., Fuqua, C., Brinkac, L., Lewis, M., Johri, S., Weaver, B., Pal, G., Eisen, J., Rahe, E., Sheldon, W. M., Ye, W., Miller, T. R., Carlton, J., Rasko, D. A., Paulsen, I. T., Ren, Q., Dougherty, S. C., DeBoy, R. T., Dobson, R. J., Durkin, A. S., Madupu, R., Nelson, W. C., Sullivan, S. A., Rosovitz, M. J., Haft, D. H., Selengut, J., and Ward, N., 2004, Genome sequence of *Silicibacter pomeroyi* reveals adaptations to the marine environment, *Nature (London)* **432**:910 913.

Novak, R. T., Gritzer, R. F., Leadbetter, E. R., and Godchaux, W., 2004, Phototrophic utilization of taurine by the purple nonsulfur bacteria *Rhodopseudomonas palustris* and *Rhodobacter sphaeroides*, *Microbiology (Reading)* **150**:1881 1891.

Rabus, R., Ruepp, A., Frickey, T., Rattei, T., Fartmann, B., Stark, M., Bauer, M., Zibat, A., Lombardot, T., Becker, I., Amann, J., Gellner, K., Teeling, H., Leuschner, W. D., Glöckner, F. O., Lupas, A. N., Amann, R., and Klenk, H. P., 2004, The genome of *Desulfotalea psychrophila*, a sulfate reducing bacterium from permanently cold Arctic sediments, *Environ. Microbiol.* **6**:887 902.

Reichenbecher, W., Kelly, D. P., and Murrell, J. C., 1999, Desulfonation of propanesulfonic acid by *Comamonas acidovorans* strain P53: evidence for an alkanesulfonate sulfonatase and an atypical sulfite dehydrogenase, *Arch. Microbiol.* **172**:387 392.

Rein, U., Gueta, R., Denger, K., Ruff, J., Hollemeyer, K., and Cook, A. M., 2005, Dissimilation of cysteate via 3 sulfolactate sulfo lyase and a sulfate exporter in *Paracoccus pantotrophus* NKNCYSA, *Microbiology (Reading)* **151**:737 747.

Ruff, J., Denger, K., and Cook, A. M., 2003, Sulphoacetaldehyde acetyltransferase yields acetyl phosphate: purification from *Alcaligenes defragrans* and gene clusters in taurine degradation, *Biochem. J.* **369**:275.

Shimamoto, G. and Berk, R. S., 1980, Taurine catabolism II. Biochemical and genetic evidence for sulfoacetaldehyde sulfo lyase involvement, *Biochim. Biophys. Acta* **632**:121 130.

Styp von Rekowski, K., Denger, K., and Cook, A. M., 2005, Isethionate as a product from taurine during nitrogen limited growth of *Klebsiella oxytoca* TauN1, *Arch. Microbiol.* **183**:325 330.

Weinitschke, S., Styp von Rekowski, K., Denger, K., and Cook, A. M., 2005, Sulfoacetaldehyde is excreted quantitatively by *Acinetobacter calcoaceticus* SW1 during growth with taurine as sole source of nitrogen, *Microbiology (Reading)* **151**:1285 1290.