pure rare earth compounds, $DyCoO_3$, $NdCoO_3$, and $LaCoO_3$, are strikingly similar to each other and to $RCoO_3$ where R (*6*) is the natural rare earth mixture as mined.

We suggest, therefore, that the unseparated rare earth oxides can be used to make a cheap auto exhaust oxidation catalyst.

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Passive Ventilation in Benthic Annelids?

Vogel and Bretz (*1*) have recently suggested that passive ventilation is of respiratory importance to burrowing intertidal worms, and that the spontaneous rhythmic bursts of irrigation activity so widespread in the group serve some sort of function in burrow maintenance. They propose that boundary effects of water currents flowing past burrow openings result in the passive flow of water through the burrow, which serves to furnish oxygen to the animal. They suppose that the active pumping of water through the burrow may serve to maintain such burrow structures as the head shaft by preventing the excessive packing of the sandy sieve that fills it. We would like to point out that existing knowledge of ventilatory behavior in annelids does not support these arguments, that annelid pumping activity clearly serves a respiratory function, and that passive flow can be of little importance to annelids.

First, in the cases examined (*2*), the oxygen consumption of worms in tubes parallels exactly the spontaneous irrigation activity recorded and observed when the animals are in their tubes or burrows. There is a low rate of oxygen consumption during rest and a rapid uptake during bursts of burrow irrigation. The increased oxygen uptake is greater than that required to reoxygenate the small volume of water remaining in the tube at the onset of pumping activity, and bursts of activity last for much longer periods than would be required to flush the burrow (*2*, *3*). During the pumping, the organs of gas exchange are ventilated and the respiratory pigment is oxygenated (*2*). Moreover, a respiratory function has been demonstrated directly by measurement of a lower oxygen content in the excurrent than in the incurrent stream generated by spontaneously irrigating worms (*4*). And the surprisingly high velocities of currents generated by annelids suffice to account for their respiratory needs (*2–5*).

Although spontaneous rhythmic irrigation in annelids was originally characterized from a closed system (*6*), several more recent studies have been made of animals placed in moving seawater (*3*, *5*), and one very ingenious investigation on *Arenicola marina* was conducted in the worm's natural habitat (*7*). Therefore, both active pumping and its cessation occur in the presence of water current flow overhead.

Second, when *Arenicola* [the example cited by Vogel and Bretz (*1*)] is resting in its burrow between bursts of pumping activity, its body shortens and thickens, effectively plugging the burrow (*8*) and thus making passive flow by any mechanism impossible. The same is true of *Glycera dibranchiata* (*2*), several species of *Clymenella* (*3*), *Amphitrite ornata,* and many other sedentary species. The ventilation current in infaunal worms is often generated by the progression of peristaltic waves of muscular contraction in either direction, with the direction dependent upon the orientation of the worm. Distortion of the body is usually restrained by the walls of the tube or burrow, and hence the action of pumping also occludes the burrow along the length of the shaft. Although passage of peristaltic waves ceases during rest, local contractions of the longitudinal muscles are sustained, thus maintaining the points of contact when the worm is not physically plugging the whole diameter of the burrow.

A different mechanism of generating currents is employed by errant species, but the habit of most of these worms violates the physical requirements mentioned by Vogel and Bretz (*1*). Indeed, epifaunal quill worms of the genus *Hyalinoecia* actively prevent passive flow through their tubes by constructing one-way valves at either end (*9*).

Perhaps most important, it is not clear that passive ventilation would be quantitatively important for any aquatic animal that actively generates currents. Although the mechanism may be applicable to sponges and other groups, its significance must be assessed on the basis of its magnitude. In any event, it is clearly inapplicable to annelids. The respiratory function of irrigation activity has been demonstrated, and resting worms plug their burrows and prevent passive flow through them.

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We agree with Hoffman and Mangum that for any species the significance of passive ventilation will be evident only upon determination of the magnitude of the phenomenon and its energetic benefits. We plan to make such measurements on one or more polychaetes and hope that other investigators will do so also.

Any reduction in work should provide sufficient selective advantage to lead us to expect that a species whose habits meet the physical requirements will capitalize on passive ventilation. Furthermore, demonstration that active pumping can be adequate to meet an animal's respiratory needs does not constitute evidence that the animal cannot benefit from passive ventilation. All of the studies cited by Hoffman and Mangum were carried out under experimental conditions which completely precluded passive ventilation. Even Krüger (*1*) employed a bell jar over one end of the *Arenicola* burrow. We do not question any of these results but merely suggest that another phenomenon of potential significance may have inadvertently been overlooked.

Does *Arenicola* plug its burrow between bursts of pumping? Wells (*2*), commenting on his observation of a worm in a glass tube beneath still water, stated only: "In the relatively quiet intervals between outbursts, the worm was lying in the curved part of the U, with its head toward the float and its body rather short and thick." He further noted that "worms appear to be more active between the bursts when in sand than they are in glass tubes." For worms that make single-ended burrows, however, passive ventilation can be disadvantageous; viscous entrainment at the aperture will reduce the pressure in the burrow and may draw water from anoxic sediments into it. Thus plugging of burrows might be expected in some of the other species mentioned by Hoffman and Mangum.

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Lipid Biosynthesis and Increase in Isovaleric Acid of Plasma

Tanaka *et al.* (*1*) reported that the administration of hypoglycin A, a causative agent of Jamaican vomiting sickness, results in an increase in isovaleric acid in the plasma of rats. They suggested that the increased levels result from an overloading of the glycine conjugating system. In isovaleric acidemia, a disease with parallel symptoms, the increase in isovaleric acid is explained in terms of the inhibition of isovaleryl coenzyme A (CoA) dehydrogenase (*2*).

However, an additional pathway involving lipid biosynthesis may also be operative. Horning *et al.* (*3*) have shown that in a partially purified soluble enzyme system from rat adipose tissue isovaleryl CoA is readily condensed with malonyl CoA in the biosynthesis of long-chain iso acids. Under the experimental conditions, the isovaleryl CoA is incorporated at a rate almost comparable to that of acetyl CoA. A notable example of isovaleric acid "detoxification" is found in porpoise acoustic tissues where large amounts of lipids containing isovaleric acid and long-chain iso acids are deposited (*4*). Accordingly, enzymes capable of incorporating isovaleryl CoA into lipids are present in normal mammalian tissues. The fact that only small amounts of the iso acids are usually found simply reflects the low concentrations of isovaleryl CoA present (*3*). Therefore, we suggest that blocking of incorporation of the isovaleryl CoA in lipid biosynthesis may be an additional factor in the accumulation of isovaleric acid in the plasma of patients inflicted with the Jamaican vomiting sickness or isovaleric acidemia.

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We have demonstrated that the accumulation of the isovaleryl moieties (isovalerate + isovalerylglycine) seen in both isovaleric acidemia (*1, 2*) and hypoglycin-treated rats (*3, 4*) is due to the depression of leucine oxidation at the stage of isovaleryl CoA dehydrogenase. Thus, isovaleryl CoA is not oxidized further (*1, 3*); but is alternatively conjugated with glycine (*2–4*). Consequently, a large amount of isovalerylglycine is excreted in urine in both cases (*2, 4*). When the amount of isovaleryl CoA exceeds the capacity of the glycine acylating system, the excess would be hydrolyzed, resulting in a release and accumulation of free isovaleric acid (*2, 4*). The derangement of leucine metabolism in isovaleric acidemia is identical to that in hypoglycin-treated rats. The basic difference is that the former is due to the genetic deletion of isovaleryl CoA dehydrogenase, whereas the latter is induced by inhibition of the same enzyme.

Branched long-chain fatty acids are minor components in mammalian lipids (*5*). It has been known since 1823 that fats from some tissues of dolphins and porpoises are unique in containing a considerable proportion of isovaleric acid (*6*). Isovaleric acid has never been identified as a component of lipid from other mammals.

There is no reason therefore to speculate that blocking of incorporation of isovaleryl CoA into lipid biosynthesis is another contributing factor to the pathogenesis of the two diseases. As a rule, inborn errors of metabolism are usually the result of single enzyme deletions, and to postulate a deletion of two enzymes, without evidence, seems unnecessary.

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