Fatal methane and cyanide poisoning as a result of handling industrial fish: a case report and review of the literature

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Abstract
The potential health hazards of handling industrial fish are well documented. Wet fish in storage consume oxygen and produce poisonous gases as they spoil. In addition to oxygen depletion, various noxious agents have been demonstrated in association with spoilage including carbon dioxide, sulphur dioxide, and ammonia. A fatal case of methane and cyanide poisoning among a group of deep sea trawler men is described. Subsequent independent investigation as a result of this case led to the discovery of cyanides as a further potential noxious agent. This is thus the first case in which cyanide poisoning has been recognised as a potentially fatal complication of handling spoiled fish. The previous literature is reviewed and the implications of the current case are discussed.

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Case report
A fatal accident occurred aboard a stern freezing trawler 40 miles off the coast of Mauritania in West Africa while engaged in pelagic trawling for fish. The vessel was close to the end of a 30 day spell of fishing and was sorting the containers held at 20°C, 35°C, and 45°C. The risk is especially high in industrial fishing because fish are stored in bulk without ice in closed spaces. Factors that aggravate fish spoilage are a relatively high temperature and lack of ventilation, and under these conditions a

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dangerous atmosphere can be produced within hours. The initial flora of North Sea fish consisted mostly of moraxella, arthrobacter, pseudomonas, flavobacterium, cytophage, and micrococcus but pseudomonas became more dominant on prolonged storage.1 Pseudomonas and hydrogen sulphide producing Altermonas putrefaciens are responsible for spoilage even though moraxella, acinetobacter, and coryneforms are present in large numbers.2 Previous reference has been made to the production of carbon dioxide, sulphur dioxide, and ammonia from rotten fish. In the present case the noxious agents appear to have been methane and cyanide. Methane is a well known product of putrefaction, but it is interesting to speculate how a potentially fatal load of cyanide can build up in a catch of fish, particularly because this is the first recognised case.

Cyanide is a secondary metabolite formed by the oxidation of glycine to hydrogen cyanide and carbon dioxide by bacteria—Chromobacterium violaceum and pseudomonas.3 Cyanide binds irreversibly with cytochrome c oxidase in both transient turnover and stable states, and as such acts as a metabolic poison.4 The rate of cyanide production increases at higher temperatures, with maximum cyanogenesis occurring at 25–30°C.5 Hydrogen cyanide produced by Pseudomonas aeruginosa in a synthetic medium required aerobiosis.6

The simulated conditions generated by the team from the University of Bristol were likely to have closely mirrored those present in the refrigerated salt water tank on board the trawler at the time. Certainly, the speed with which the fatally injured crewmen were seen to collapse and the range of symptoms demonstrated by the survivors are in keeping with the effects of a metabolic poison such as cyanide. Previous reports of industrial incidents related to decaying fish have failed to mention cyanide as a potential toxic agent.

Pseudomonas putrefaciens is an organism found on fish producing hydrogen sulphide.7 We are aware of two other cases of caused by the inhalation of toxic fumes from decaying fish; hydrogen sulphide was presumed to be the main cause and cyanides were not found. Proteus, a much less common organism on fish, produces hydrogen sulphide8 and Pseudomonas putrefaciens is another organism found on fish that produce hydrogen sulphide.9 Hydrogen sulphide is associated with the risk of hypoxic brain damage. In both these cases of death caused by the inhalation of toxic fumes, consciousness was lost immediately. Follow up of these patients revealed various neurological abnormalities such as reduced memory, irritability, reduced motor function, reduced vibration and temperature sense, ataxia, positive rhombergs sign, considerably reduced learning and retention, and dementia. The computed tomography scan showed widened ventricles and cortical atrophy in one of the cases. Interestingly, both men were awarded a disability pension within a year of the accident. Blood sulphide may be used as an indicator of excessive hydrogen sulphide exposure but the sample has to be taken as soon as possible (not later than two hours) and analysed without delay.10 Clearly, this was not possible in this case. Hydrogen sulphide is associated with decreased activity of haem synthesising enzymes especially amino leuvalinic acid synthase and haem synthase.11

The spoiled fish in our case were oily fish and it is interesting to speculate whether white fish would spoil in a similar manner. The organisms associated with North Sea fish have previously been listed. In warmer climates, different flora predominate such as bacillus species, micrococcus, and coryneforms.12 Would the products of spoilage be different? This might be an avenue for further research.

In conclusion, a fatal case of inhalation of toxic fumes related to decaying fish is reported. The putative agents involved in this case appear to be methane and/or cyanide. Awareness of the health hazards involved in the handling of industrial fish is important, particularly for those working in the vicinity of fishing communities.

We thank the marine accident investigation branch of the Department of Transport for allowing us to publish the case and for allowing the study undertaken by the food refrigeration and process engineering research centre (PRPERC), University of Bristol as a result of this case.

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