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**Research paper**

# SULPHIDE DUST EXPLOSIONS IN MINES – $SO_2$ EXPOSURE AND RESPIRATORY EFFECTS

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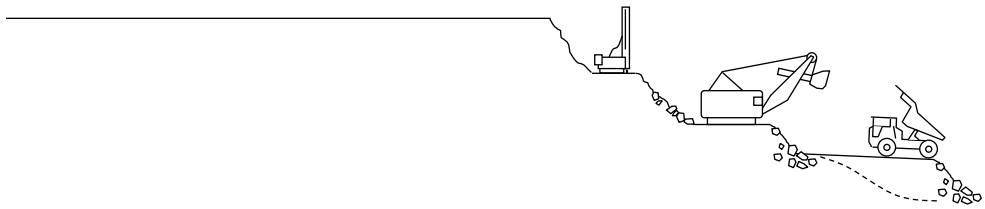
## ABSTRACT

Sulphide dust explosions and ignitions with high levels of $SO_2$ may be a problem in rich sulphide ores, but accidentally high levels of $SO_2$ have also occurred in paper mills, cellulose industry, foundries, etc. Fatal accidents and accidents with lung damage after $SO_2$ exposure have been described since the early twenties in the USA. Little is known about the long-term effects of acute $SO_2$ exposure and the clinical significance of $SO_2$ exposure is debated. The aim of this presentation is to describe some effects on the respiratory system because of high levels of $SO_2$ exposure. For obvious reasons it is impossible to measure the exact level of exposure. The lung function is vulnerable and hyper reactive after such accidents and immediate hospitalization and professional medical care and follow up is mandatory. The development and the final outcome of the lung damage can not be predicted until a significant period of time has elapsed. Codes of practice do not focus clearly on this fact. The only way to avoid such accidents in sulphide rich mines is to be sure that there are no workers in the mine during blasting operations.



## 1. INTRODUCTION

Department of Geology and Mineral Resources Engineering, NTNU, former SINTEF Rock and Mineral Engineering, University of Trondheim, has for more than 40 years carried out testing and documentation of dust, gases, radon and other air pollutants associated with mining.

The objectives here have been both an estimation of the exposure levels in sulphide dust explosions and ignitions, along with documentation of the health effects of such exposures. In addition we want to stress the importance of professional medical follow up of the workers involved.

## 2. MATERIALS AND METHODS

Estimation of exposure and exposure levels are based on data from six Norwegian sulphide ore mines, all of which are now categorized as historical because they are already closed, as well as results from similar accidents and also experimental studies. The six sulphide ore mines referred to are Grong Gruber, Folldal Verk AS (Tverrfjellet), Sulitjelma, Løkken Verk, Killingdal and Skorovas (Myran & Furuseth 1991).

Ignitions have occurred in high-grade sulphide ore deposits, but also in low-grade sulphide ores, during all types of underground blasting operations. The frequency of sulphide ignitions in these mines varied from 3 to 5 times per year and up to 1 to 3 times per week depending on the local conditions.

Ignitions have occurred in situations with:

- Content of sulphur higher than 15%. Most frequently S > 40%.
- Temperatures in the mines. 4-25º C (average 9º C).
- Relative humidity, 4-100%.
- Square areas of roadway, slope or opening. 3-65 $m^2$.
- Length of round, 1.6 to 4.2 m.

We have described two accidents, sulphide dust explosions or burns in the mining industry in Norway, at Folldal Verk AS in December 1968 and at Grong Gruber in April 1989 involving 6 (4+2) workers altogether. This material also describes the development of lung damage of the 6 workers after they figured in the accidents.

Examples of the effects of extreme $SO_2$ exposure at Folldal Verk AS in 1968 (Items 1-4) and Grong Gruber in 1989 (Items 5-6) are the following:

1. Death
2. Severe Chronic Obstructive Pulmonary Disease (COPD) and cor pulmonale
3. Moderate COPD
4. Temporary impairment
5. Severe COPD and lung transplant
6. Moderate COPD

Post accidental examinations and investigations indicate that the differences in SO2 exposures are the main reason for the differences in outcomes.

The companies' Safety Health and Environment (SHE) services had a long tradition for regular medical follow-up of their workers. This included investigations with reference to lung diseases and reduced lung function (spirometry or lung function test) and chest X-ray at the cooperative hospital's lung department. These medical examinations were performed periodically, from yearly to every third year. Chest X-ray was usually performed every third to every fifth year, depending on the workers clinical status and the exposures.

Medical treatment and follow up were also done at the regional hospitals, in Namsos and Levanger for the workers in the Grong Gruber. Similarly, the regional hospital in Elverum cooperated with the SHE service at Folldal Verk AS. MD Jan Schaanning at the University Hospital in Trondheim conducted the medical examinations of mineworker HO. MD Randi Sudbø Brandzæg and MD Thor Naustdal have done, and still do the follow-up of the mineworkers KSA and BRS respectively.

Results from 16 and 21 years of follow up are given.

## 3. RESULTS

### *3.1 Exposure*

It is impossible to measure the exact level of exposure and exposure time in such accidents, for obvious reasons. We can, however, conclude that the exposures have been extreme, based on the clinical effects and the results from measurements after similar sulphide dust explosions.

Experimentally induced sulphide dust explosions have given concentrations of $SO_2$ in the range of 300-1600 ppm (Hall 1987). There are usually also some nitrous oxides and a little $H_2S$. The highest risk of sulphide dust explosions occur when particles are less than 50 micrometer, the concentration 1.200 gram per $m^3$ (200-4.000 gram per $m^3$ ) and the sulphur concentration more than 38-40% in the dust cloud. Threshold Limit Value (TLV) in Norway is 2 ppm $SO_2$. With regard to estimation of exposure levels, see chapter 4.

The important parameters for sulphide dust explosions in pyrite mines are:

- Sulphide dust concentration:
  - 2000 g/$m^3$ (max. explosibility pressure, Folldal mine)
- Sulphide content in dust:

- > 38-40% S.

- Particle size:
  - 1-10 micron, max explosibility.
  - > 300 micron, cannot explode.
- Particle form:
  - Round particles have lower explosibility than irregular and sharp edged particles.
- Ignition energy and temperature.
  - Square area of particles: > 3500 $cm^2/g$.

### *3.2 Exposure of $SO_2$ and effects on the respiratory system.*

One of the mineworkers died in the accident in 1968, one survived with temporary impairment, one got moderate chronic obstructive pulmonary disease (COPD) and one serious COPD with secondary heart disease, cor pulmonale.

In the accident in 1989 one of the workers got moderate COPD and the other serious COPD. The last mentioned worker had a lung transplant 18 years after the accident.

#### *3.2.1 Detailed description of the post accidental development.*

Mineworker HO was born in 1938. He was 30 years of age when involved in the accident in December 1968. He was a healthy non-smoker. During and immediately after the accident he had a severe cough and he was almost blinded because of the noxious gases and the intense heat. He was weak and exhausted. Chest x-ray at the local hospital, in Tynset, revealed an infiltration in his right lung. Body temperature was 38.3 degrees C. He was treated with substantial doses of steroids and antibiotics and the situation "normalised". After 9 days he left the hospital.

There came a worsening the day after he left the hospital with severe breathing problems. Further medical follow-up was done at the Dept. of Lung Medicine, University Hospital Trondheim.

In January 1969 he still had severe dyspnoea, even when resting. His lung function had been dramatically reduced during the accident. Vital capacity (VC) was more than halved and forced expiratory volume in one second ($FEV_1$) was reduced to 25-30% of the pre accidental level.

One year after the accident it was concluded that he had a severe obstructive lung disease with pathologic distribution of flow. During the following years he often suffered exacerbations, aggravation of his obstructive lung disease with lung infections. He also developed hypertension and polycythemia as well as angina

pectoris. He died suddenly, probably because of a cardiac infarction in 1985, only 47 years of age (Furuseth & Myran 1991).

Mineworker KSA born in 1952 was 36 years of age when involved in the accident in 1989. He was a healthy non-smoker. In the following years he suffered seriously from his lung damage, with dyspnoea and exacerbations and in 2007 he had a lung transplant. His lung function was markedly reduced because of the accident with FEV1 less than 20% of pre accidental values.

Mineworker BRS born in 1955 was 33 year of age when involved in the accident in 1989. He was a healthy, daily smoker. When examined in September 2010 his lung function was still markedly reduced, especially $FEV_1$ which is more than halved compared to pre accidental values. Even though, he in March 2012 describes himself as healthy with little breathing problems. He uses no medication regularly, only beta-2 agonist on occasions.

The following lists give an overview of the consequences for the victims.

Accident in 1968:

- Worker 1- (WF)
  - died in the accident.
- Worker 2- (JB)
  - sick leave
  - continued mining
  - moderate COPD
  - died 87 years old.
- Worker 3- (SB)
  - sick leave
  - continued mining
  - 69 years old and healthy (2012).
- Worker 4- (HO)
  - hospital and re-hospitalization
  - change of work (lift operator in the mine)
  - serious COPD ($FEV_1$ 25-30% of preaccidental value) and cor pulmonale
  - died 16 years after the accident, age 47.

Accident in 1989:

- Worker 5- (KSA)
  - hospital and re-hospitalization

    - serious COPD, FEV1 10-20% of preaccidental value
    - lung transplant (2007)
    - managing well in the circumstances (2012)
- Worker 6 – (BRS)
    - hospital
    - sick leave
    - reduced lung function, $FEV_1$ 45% of preaccidental value
    - change of work, now truck driver (2012)

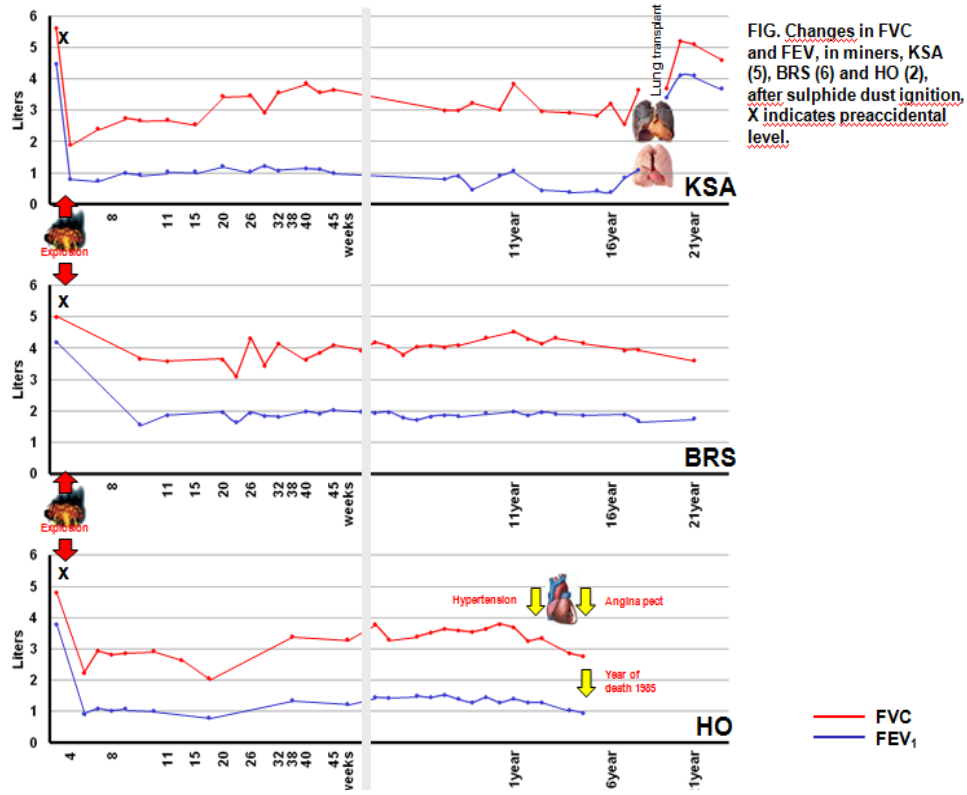
All (Workers 1-6) > bronchial hyperreactivity All (Workers 1-6) > bronchial obstruction : 1/5 moderate, 1/5 severe and 2/5 very severe COPD with pulmonary hyperinflation (small airway disease)

The actual time of exposure to the noxious gases varied for the miners involved in the described accidents. In the accident in 1989 the two workers involved suggested that they were exposed to noxious gases for 1 hour, more or less. They considered the “first cloud” from the explosion to be the most extreme with a strong smell of sulphur and immediate dyspnoea and a feeling of choking. They tried to breathe through their clothes.

In the accident in 1968 the worker who died had the most extreme exposure. The worker who developed serious lung damage was heavily exposed. The two who managed without measurable lung damage were situated at the longest distance away from the accident, but they also felt the discomfort of the sulphur dioxide exposure. Worker SB described, in March 2012, that he and his colleague JB reacted to "almost everything" the following weeks and months with symptoms of coughing and dyspnoea. These after accidental symptoms indicate bronchial hypersensitivity reaction secondary to the sulphur dioxide exposure.

It is important to emphasize that although the two mineworkers involved in the accident in 1989 were exhausted, they did not look "seriously ill" the first hours after the exposure. They themselves had to insist on hospitalisation and arriving hospital insist on staying there. There came a gradual worsening during the following days and weeks, from one to more than 3 weeks. One of the workers, KSA, said (June 2011) that post accidental day 23 was the worst. Despite treatment with diuretics and prednisolone he could hardly move the second and third week after the accident. The following year was hard with dyspnoea on minor exertion, exacerbations and hospitalizations.

In all three cases FVC is significantly reduced, but less compared to the loss in $FEV_1$. FVC also tend to improve a little, in contrast to $FEV_1$ (fig. 1).




**Figur 1.** Changes in lung function, forced vital capacity (FVC) and forced expiratory volume in one second ($FEV_1$) in miners KSA, BRS and HO after sulphide dust ignition. X shows pre accidental lung function.

The lung damage and the lung function loss because of the accident and the $SO_2$ exposure are considerable. The involved workers lives were dramatically changed and their possibilities of work and physical activity were turned upside down. Although the outcome is different, there are similarities in the development of the lung diseases. Mineworker HO died 17 years after the accident due to his lung and secondary heart disease. Mineworker KSA has had a lung transplant 8 years after the accident. His lung function was, at the time of the lung transplant, more than 80% reduced compared to pre accidental values. Even mineworker BRS, who describes himself as healthy with little breathing problems, has had his lung function halved compared to the values measured before the accident.

## 4. DISCUSSION

$SO_2$ is highly irritative. In contact with moist epithelium it is hydrated and oxygenated to sulphuric acid. This may give rise to serious damage to the epithelial lining of the airways and bronchial tree.

Medical literature indicates 400 ppm to be a deadly concentration of $SO_2$. 40 ppm is said to give immediate dyspnoea and also reduced vision. Concentrations from 6 to 12 ppm give upper respiratory tract irritation. It is usually possible to smell $SO_2$ in concentrations from 0.3 to 1,0 ppm. As much as 99% of the $SO_2$ is said to be absorbed in the upper respiratory system. But, as we have shown, $SO_2$ may also result in damage in the smaller airways or peripheral respiratory bronchioles.

Low concentrations of $SO_2$ (0.2-0.6 ppm) is usually used in experimental studies. But even in these studies changes are seen in lung function and macrophage activity although they are reversible within 1 to 3 days (Linn 1983).

Some case reports following accidents with high exposure to $SO_2$ indicate bronchial hypersensitivity reactions, reversible and irreversible bronchial obstruction years after the accident (Härkönen 1983). Other reports show little or no long-term harmful effects (Skalpe 1964).

Deaths occurring within a few hours of exposure are due to pulmonary oedema, while deaths due to bronchiolitis obliterans are described to occur three to five weeks later (Galea 1964).

In a 4 year follow-up of 7 mineworkers accidentally exposed to high levels of $SO_2$ in an explosion in a pyrite mine in Finland in 1977 (Härkönen 1983) the greatest decreases in lung function (FVC, $FEV_1$, and MMEF) are described one week after the accident, after which all these parameters improved without reaching pre accidental levels. Reversible bronchial obstruction was still present in three of the seven, 4 years after the accident. They all complained of breathlessness during pronounced effort. However, a 13 year follow-up of 6 of the 7 mineworkers already mentioned showed that all six mineworkers re-examined in 1990 still had markedly decreased excertion tolerance and they described this to be the most notable negative effect of the accident (Piirilä 1996).

Piirilä et al. (1996) concluded that exposure to $SO_2$ with acute inflammatory obstruction caused as a sequela, obstructive impairment of ventilatory function and permanent bronchial hyperreactivity. Of the six, four also showed symptoms of chronic bronchitis.

In an industrial accident Charan et al. (1979) described 5 previously healthy persons who were acutely exposed to very high concentrations of sulfur dioxide. The two with the highest exposure died immediately. Histologic examination of the lungs revealed extensive sloughing of the mucosa of large and small airways along with hemorrhagic alveolar edema. The 3 survivors were evaluated with pulmonary function tests performed at regular intervals. One subject subsequently developed symptomatic severe airway obstruction unresponsive to bronchodilators. Another subject developed asymptomatic mild obstructive and restrictive disease, and the third subject continued to be asymptomatic with normal pulmonary function tests. This report serves to document the histologic features of fatal exposure to sulfur dioxide and stresses the need to follow parameters of pulmonary function in the nonfatal cases.

Our results support these findings with outcomes ranging from hardly any impairment to severe COPD and death.

Workers involved do not necessarily look seriously ill after such accidents. Reduction in lung function may develop fast, deteriorate during the following days or weeks and end up in serious and chronic lung disease as well as death.

Although we cannot exclude individual susceptibility we believe the different outcomes to be due mainly to differences in exposure level and duration of exposure. All the mineworkers involved were in good health and physically active before the accident.

Based on the clinical signs of the involved workers, the results from sulphide dust explosion studies and experimental studies one might estimate the exposure levels in the two described accidents to be between 6 and 400 ppm. The nature of these accidents, however, makes such estimations insecure. This, together with the fact that these workers may not necessarily look ill, stresses the need of immediate hospitalisation and thorough medical follow up for sufficient time.

Since the beginning of the last century a lot of literature and code of practice have described the harmful effects of sulphur dioxide, exposure levels and clinical signs as well as the importance of preventive actions to avoid sulphur fires and explosions. Some additional interesting examples are Gardener et al. (1926), Hall et al. (1987) and Hunter (1990).

The long term effects of sulphur dioxide exposure are debated. We believe this mainly to be due to the difficulties and the unreliability of exposure measurements together with inadequate medical follow up. As far as we have seen code of practice seldom focus on the need of thorough, professional and sufficient time medical follow up of workers exposed to sulphur dioxide (Occupational Safety and

Health Services, 1993). One code of practice naturally expects the occupational physician to initiate the necessary measures (National Institute for Occupational Safety and Health, 1978), others are more specific (Badenhorst 2007).

## 5. CONCLUSIONS

The workers involved in the two described accidents, sulphide dust explosions, were exposed to very high levels of $SO_2$, probably some place between 6 and 400 ppm $SO_2$. These estimations are naturally insecure. The same can be said about estimations of the exposure time. Clearly, the exposure level and/or the exposure time have been substantial.

The obstructive ventilatory impairment, the bronchial hyper reactivity and the chronic obstructive bronchitis seen after such accidents may develop fast and may give serious and permanent disablement.

Very often there comes a gradual worsening some time after the accident, in the cases we have described this happened during the first weeks and up till more than 3 weeks after the accident.

Cautious and professional medical care after exposure to high levels of sulphur dioxide is mandatory. Workers involved do not necessarily look seriously ill after such accidents. Reduction in lung function may develop fast and may end up in serious and chronic lung disease. Confirmed cases should receive immediate hospital treatment and thorough medical follow up for sufficient time.

We therefore advise these codes of practice to focus more on the importance of long-term medical follow up.

We also conclude that the only safe place for workers in sulphide rich mines during blasting operations is outside the mine.

As a consequence of the accident in 1968 blasting operations in such mines were regulated. It was no longer permitted to blast bigger rounds whilst people were in the mine.

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## REFERENCES

Badenhorst, C.S., 2007: Occupational health and safety risks associated with sulphur dioxide. The Journal of The Southern African Institute of Mining and Metallurgy, vol. 107:299-303.

Charan, N.B., Myers, C.G., Lakshminarayan, S., Spencer, T.M., 1979: Pulmonary injuries associated with acute sulphur dioxide inhalation. Am. Rev. Respir. Dis., 119:555-560.

Furuseth, K., Myran, T., 1991: Svoveldioksyd og lungeskade. Eksposisjon og sykdom belyst med eksempler. Helse og miljø i bergindustrien. Delrapport 8. STF36 F911103.

Galea, M., 1964: Fatal Sulphur Dioxide Inhalation. Case Report. Canad. Med. Ass. J. Aug. 15, vol. 91.

Gardener, E.D., Jones, G. W., Sullivan, J.D., 1926: Gases from blasting in heavy sulphides. U.S. Department of the Interior, Bureau of Mines.

Hall, A., McHaina, D.M., Botsfjord, J.D., Rohr, S., 1987: Sulphide Dust Explosion Studies at H-W-Mine of Westmin Resources Ltd. Proc. of 4th Mine Vent. Symp.

Hunter, P.R., 1990; An underground explosion at Doornkop No 1 Shaft. Journal of the Mine Vent. Soc. of S.A.

Hãrkõnen, H., 1983; Long-term effects of exposure to sulphur dioxide. Am. Rev. Respir. Dis. 128:890

Linn, W.S. , Venet, T.G., Shamoo, D.A., Valencia, L.M., Anzar, U.T., Spier, C.E., Hackney, J.D., 1983: Respiratory effects of sulphur dioxide in heavily exercising asthmatics. A dose-response study. Am Rev Respir Dis 127 (3): 278-283.

Myran, T., Furuseth, K., 1991: Kisstøveksplosjoner. Risiko. En teknisk og medisinsk vurdering. Helse og miljø i bergindustrien. Delrapport 5. STF36 A91073.

National Institute for Occupational Safety and Health, 1978: Occupational Health Guideline for Sulphur Dioxide. U.S. Department of Health and Human services. U.S. Department of Labour.

Occupational Safety and Health Services, 1993:Approved code of practice for the prevention of sulphur fires and explosions. Health and safety in employment act 1992. Department of Labour. Wellington. New Zealand.

Piirilä, P.L., Nordman, H., Korhonen, O.S., Winblad, I., 1996; A thirteen-year follow-up of respiratory effects of acute exposure to sulfur dioxide. Scandinavian Journal of Work, Environment and Health. 22(3): 191.196.

Skalpe, I.O., 1964: Long-term Effects of Sulphur Dioxide Exposure in Pulp Mills.Br J Ind Med; 21(1): 69-73.