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Inhalation of ammonium nitrate fuel oil explosive (ANFO): and possible concomitant exposure

EDITOR,—Donoghue¹ reports on respiratory
symptoms and rhonchi in a miner after expo-
sure to ammonium nitrate fuel oil explosive
(ANFO). As diesel fuel is the most commonly
used fuel in ANFO the vapour he refers to
might be components of diesel fuel. He
excludes concomitant exposure to nitrogen
dioxide because the inhalation occurred
before any explosion took place. Although
diesel powered machines are commonly used
in underground work he does not discuss
possible exposure to diesel exhaust. I have
measured up to 15 ppm nitrogen dioxide
during construction of a tunnel where the
only known source was diesel exhaust. Expo-
sure to such high concentration may contrib-
ute to respiratory symptoms and rhonchi.
Therefore nitrogen dioxide should not be
excluded as a concomitant causative factor.
In a study of the contribution of gases from
diesel exhaust and from the blasting cloud
caused by the ANFO explosive during
excavation of a tunnel, diesel exhaust contrib-
uted most to the total amounts of nitrogen
dioxide in the tunnel.²

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nitrate fuel oil explosive (ANFO). *Occup Environ Med* 1998;55:144.
- 2 Søstrand P, Lian K, Myran T. The contribution
of inorganic gases from diesel exhaust and from
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Occupational asthma due to amylase

EDITOR,—In a letter to you,¹ Hendrick points
out my inadvertently overlooked report in
The Lancet on allergy to α -amylase and
papain,² and makes generous reference to my
other work on enzymes.

For clarification, I should point out that the
evidence, supported by the findings with
amylase, that sensitisation and consequential
symptoms may occur from proteases inde-
pendently of proteolytic activity, must not
obscure the fact that proteolytic mechanisms
may cause other clinical and subclinical
effects. As well as skin irritation, non-
sensitised people may experience epistaxis or
haemoptysis, whereas rhinorrhoea or asthma
are more likely to be due to allergy.
Propensity to cause proteolytic effects varies
between different proteases, and susceptibil-
ity to such effects varies between people.³ I
think I have experienced such effects myself;⁴
and possible long term consequences have
been described.⁵

Although he concurs with the use of skin
prick tests as an index of sensitisation, Kend-
rick expresses reservations about my evidence
of causality in respect of chest symptoms
from α -amylase. This is understandable if I
relied solely on his condensation of an already
condensed report. Although my report de-
rived from a comprehensive ongoing investi-
gation, I had hoped I had summarised
sufficient information to make my point.

His reservations seem to derive from the
fact that papain was also handled in the
workplace, and that some of those sensitised
to α -amylase were also sensitised to papain.

At this factory papain and α -amylase were
handled in pure form, seldom, and at
different times and places. The association
between handling one or other material and
the development of symptoms was clear cut,
and because of the short period of handling,
with intervals of at least a month between
these periods, there was time for symptoms to
regress between exposures.

I had already validated a skin prick test for
papain sensitivity,⁶ and was able to use the
same test material for papain, and a similar
one for α -amylase. Positive prick test findings
seemed to confirm the specificity and likely
mechanism of the typically asthmatic symp-
toms from each enzyme.

Had I had any reasonable doubt as to cau-
sality I would not have published the
warning. Happily, subsequent reports, in-
cluding the excellent one by Aitkin *et al*,⁷ of
which Hendrick is a coauthor, which in-
cluded inhalation challenge tests with fungal
 α -amylase,⁷ have endorsed my conclusions
and added further knowledge.

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lase. *Occup Environ Med* 1998;55:361.
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inhalation of enzyme dusts. *Ann Occup Hyg*
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- 4 Flindt MLH. Biological miracles and
misadventures: identification of sensitisation
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- 5 Musk AW, Gandevia B. Loss of pulmonary elas-
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amylase. *Occup Environ Med* 1997;54:762–4.

Health of children born to medical radiographers

EDITOR,—There are increasing concerns that
parental workplace exposure to potentially
hazardous agents could affect the health of
future offspring. In considering this, Roman
*et al*¹ have developed a questionnaire based
method for collecting data on reproductive
outcome and child health which has been
applied to a study population comprising
(predominantly female) members of the Col-
lege of Radiographers. I have recently had
cause to revisit their report of this work in
more detail and have several comments.

The study relies on postal questionnaires for
details of adverse pregnancy outcomes, major
congenital abnormalities, and malignancies,
but only reports of cancer were validated by
reference to national registration schemes and
medical records. Comparisons with national
cancer registration rates for England and

Wales and congenital malformation rates
derived from data compiled by the Liverpool
Congenital Malformation Registry showed lit-
tle evidence to suggest an increased risk for
cancer or for major congenital malformations.
Within specific systems no excess relative risk
was evident for Down's syndrome, but two
significantly increased relative risks were
found—for "other musculoskeletal" malfor-
mations and for "chromosomal anomalies
other than Down's syndrome"—both domi-
nated by adverse outcomes reported by female
radiographers. My particular interest is with
the group of six cases of chromosome anom-
alies other than Down's syndrome.

Four cases of Turner's syndrome were
reported by female radiographers, two being
diagnosed before birth and the pregnancies
terminated. The Turner phenotype, recog-
nised in live born infants, is characterised by
monosomy X and has a birth incidence in
females of 1/2000–1/5000.² However, the
frequency of monosomy X at conception is
much higher, occurring in 1%–2% of all clini-
cally recognised pregnancies.³ Over 99% abort
spontaneously, 70% of these between 11 and
14 weeks gestation.⁴ The incidence of Turner's
syndrome will vary considerably with different
stages of pregnancy and this has implications
for the calculation of expected numbers of
cases. When evaluating Turner's syndrome in
relation to aetiological influences it is impor-
tant to have accurate information on the
timing and method of diagnosis, and compar-
isons must be made with appropriate registry
data. In around 80% of cases the X chromo-
some present is maternally derived⁵ and there-
fore, by contrast with most cases of Down's
syndrome, an error in meiotic non-disjunction
cannot be attributed to the mother. Of those
diagnosed after birth, some are mosaics with a
normal 46 XX cell line as well as a 45 X line or
a cell line with one normal and one abnormal
X chromosome, and it has been suggested that
mosaicism increases the likelihood of survival
during pregnancy.³ Such mosaics are assumed
to have arisen post-zygotically. Turner's syn-
drome is, therefore, most likely to occur due to
an error in non-disjunction arising either dur-
ing spermatogenesis or after fertilisation, and
the origin of the error can often be determined
by undertaking cytogenetic and molecular
studies. Consequently, it is unlikely that
maternal preconceptional exposure is relevant
to the occurrence of the cases of Turner's syn-
drome reported to Roman *et al*¹ although it is
possible that events immediately after concep-
tion could be implicated in the origin of any
with a mosaic karyotype. It is unfortunate that
Roman *et al*¹ provide no karyotypic data nor
information on whether the mothers were
working as radiographers when they conceived
as this would have assisted in the interpretation
of this association.

Two further pregnancies with chromosomal
abnormalities were described—a gross chro-
mosomal anomaly reported by a female
radiographer and a trisomy 17 reported by a
male radiographer—both of which were termi-
nated. Referral to results of chromosomal
studies would have provided karyotypes and the
possibility of the gross chromosomal anomaly
being the result of a familial rearrangement
could then have been explored. Trisomy 17 is
rather a surprise as this is considered incom-
patible with embryo development⁶ and has
not, to my knowledge, been detected by
antenatal diagnosis. There must be a
possibility that this information is incorrect.

Aetiological mechanisms must be consid-
ered when assessing the biological plausibility