How Dangerous is Methyl Ethyl Ketone (MEK)?

**Corresponding Author:**
Dr. Simon B Thompson,
Associate Professor, Psychology Research Centre, Bournemouth University, BH12 5BB - United Kingdom

**Submitting Author:**
Dr. Simon B Thompson,
Associate Professor, Psychology Research Centre, Bournemouth University, BH12 5BB - United Kingdom

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Author(s): Thompson S B

Abstract

Methyl ethyl ketone (MEK) is an organic solvent used throughout the paint and print industry. It is listed as a hazardous chemical by the United States Environmental Protection Agency but the neuropsychological and neuropathic effects from exposure are reported variously. Potentially, debilitating effects on occupational workers has economic as well as welfare issues; hence, there is a need to recognise and standardise assessment procedures in industry and in clinical neuropsychology.

Introduction

A comprehensive review on Methyl ethyl ketone (MEK) and the implications of exposure by workers has been provided by Thompson (2010). There is good reason for considering the effects of this chemical not only because of the welfare of occupational workers but also because of a growing number of litigation issues arising. In the United States (US), production of MEK exceeds $1.5 million annually. However, there has not been any particular focus until recently on addressing the fact that this is a widely used chemical and that this may have detrimental effects on workers exposed for long periods or at high exposures. It is acknowledged that this is not always the case in every application and that some industries have good records of safety for workers and good procedures for using potentially hazardous chemicals.

Methyl ethyl ketone is configured as CH₃COC₂H₅ or C₄H₈O (CEPA, 1999) (Illustrations 1 & 2, Wikipedia, 2011). It is also known as 2-butanone (USEPA, 1999), methyl acetone, ethyl methyl ketone, and methyl propanone. It is characterised as a colourless liquid that has a sweet or sharp, and fragrant acetone-like odour. Sometimes, it is described as minty with an irritating and unpleasant odour; and has an odour threshold (at which it may be detected) of about 16 parts per million (ppm) (Garcia, 2008). However, the current threshold limit value for occupational exposure is 200 ppm (590 mg/m³).

Discussion & Recommendations

Workers exposed to MEK at air concentrations of 3 – 6 ppm (10 – 175 mg/m³) developed blood MEK levels of 0.8 – 9.6 mg/L, averaging 2.6 mg/L with the blood/breath ratio averaging 116 and the blood/environmental air ratio averaging 35 (Perbellini, Brugnone, Mozzo, Cocheo, & Caretta, 1984). Exposure of volunteers to average MEK air concentrations of 100 or 200 ppm for 4 hours resulted in average peak MEK levels of 1.9 and 3.5 mg/L, respectively, at the end of the exposure; the levels had declined to 0.5 and 1.0 mg/L, respectively, by 1.5 hours after the end of the exposure (Dick, Brown, & Stezer, 1988).

Metabolism studies in animals has revealed that MEK is metabolised to 2-butanone, 3-hydroxy-2-butanone and 2,3-butanediol (DiVincenzo, Kaplan, & Dedinas, 1976). In humans, about 0.1% of an absorbed dose is excreted and unchanged in urine with a similar amount present as 3-hydroxy-2-butanone. Miyasaka and colleagues (1982) have reported concentration levels of MEK and this metabolite in workers exposed to air levels of MEK averaging 34 ppm (101 mg/m³). Kawai, Zhang and Takeuchi (2003) have predicted a urinary MEK level of 6.7 mg/L following an 8 hour exposure to 200 ppm.

Important in these studies is a consideration of the toxicity of MEK and the effects this has on the human body’s metabolism and central nervous system. It is known that direct air exposure to MEK causes nose and throat irritation at concentrations of 100 ppm, eye irritation at 200 ppm, headache at 300 ppm, and weakness and even paresthesias at 300 – 600 ppm. Higher levels lead to greater degrees of control nervous system depression, including confusion, loss of coordination and drowsiness. However, it is thought that MEK by itself does not cause peripheral neuropathy in animals or humans, but it does markedly potentiate the ability to cause that condition, even in small amounts according to the work of Altenkirch, Stoltenburg and Wagner (1978). Anecdotal evidence comes from Price, D'Alessandro and Kearney (1994) who report on a man who intentionally ingested 240 mL of a solution containing MEK and methanol. He was successfully treated with ethanol infusion and hemodialysis and his blood contained 1240 mg/L MEK and 240 mg/L 2-butanol, as well as 2020 mg/L methanol.
Assessment

Determination of the concentration of MEK can be made by analysing the blood, breath or urine of persons, particularly exposed to air borne MEK. Flame-ionisation gas chromatography (Chou, Shih, & Chen, 1999; Kezic, & Monster, 1988) or liquid chromatography (van Doorn, de Cock, Kezic, & Monster, 1989) has been particularly useful in chemical analysis.

In terms of assessing neuropsychological impact of MEK exposure by workers, Thompson (2010) has recommended the following neuropsychological tests: (1) General and Personal Orientation (Thompson, 2006); (2) Wechsler Adult Intelligence Scale (Wechsler, 1981); (3) National Adult Reading Test (Nelson, 1991); (4) Benton Visual Retention Test (Benton Sivan, 1991; Thompson, Ennis, Coffin, & Farman, 2007); (5) Wechsler Logical Memory sub-scale (Wechsler, 1988); (6) Trail Making Test (Corrigan, & Hinkeldey, 1987; Gaudino, Geisler, & Squires, 1995); (7) Rey-Osterreith Complex Figure Test (Rey, 1941; Osterreith, 1944); (8) Star Cancellation Task and Single Letter Cancellation Task; (9) Controlled Oral Word Association Test (Loonstra, Tarlow, Sellers, 2001); (10) Paired Associates Test; (11) Hospital Anxiety and Depression Scale (Zigmond & Snaith, 1983).

Assessing neuropsychological consequences of exposure to MEK has partly arisen from the demands of litigants in pursuance of suits against agricultural employers such as in organophosphate litigations (eg conventional “sheep-dipping” operations, particularly in the United Kingdom). Techniques used in neuropsychology have been very productive in providing a functional profile of neuropsychological deficits arising from occupational exposure, especially in respect of long-term use and exposure. Indeed, in litigation suits, neuropsychology has been helpful in deciding who is genuine (Thompson, 2003) especially since neuroanatomical facts tend not to falsely present or mask the effects of deception.

Whether the toxic effects of MEK are caused directly by a one-step process or indirectly by a degradation product of the chemical or even by second or subsequent solvents created by or acted on, the toxic effect can still occur as a result of the presence of MEK. Therefore, it makes no difference whether the toxic chemical was a result of chemical reactions. The fact remains that presenting a profile of deficits of workers exposed to MEK will help towards their treatment and some restoration of recovery, as well as presenting facts in assisting litigation or during the process of compensation.

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Illustrations

Illustration 1

Chemical Structure of Methyl Ethyl Ketone
Illustration 2

Methyl Ethyl Ketone (Schematic Structure)
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