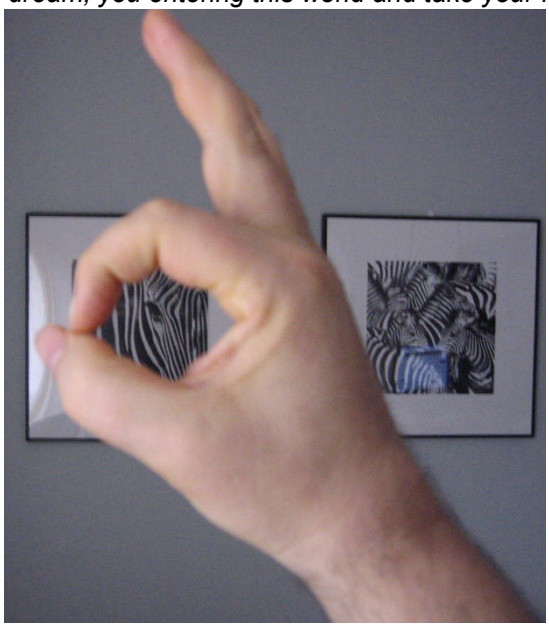
Just like a dream, you entering this world and take your first breath



The Hypo-Thesis of cognition – A Central command in emergency light
In this hypothesis I will show a possible reactive agent in the etiology of Schizophrenia This first step is showing a map of what CS2 are capable of. The next step will look into earlier steps in the etiological map of the etiology of Schizophrenia, the preconditioning.
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Corresponding Author's Institution: CAC-Hålet Theory
First Author: Pär-Olof Wassén

In Short ...
1/ There is a substance* known to cause some of the symptoms in Schizophrenia
2/The group of persons with Schizophrenia** does have it, other usually don't
3/ There might be a connection with, the reactive phase of Schizophrenia etiology and the substance.
The Substance is: Carbon Disulfide or CS2

* CS2 Intro at ADHD och ADD.

**Clin Pathol 1993;46:861-864
Increased pentane and carbon disulfide in the
breath of patients with schizophrenia

CAC-Hålet theory The reactive agent
-Showing what hypotetically could be a reactive factor in the etiology of Schizophrenia
The thermodynamics of Homo Sapiens largely rely on old CODH/ACS constructions for TCA, carbon to carbon transfer, glucose and lipid metabolism. It is shown in bacterial CODH/ACS that CS2 can alter the thermodynamics of the process by tunnel gating in centre A. (9) Themechanisms in Homo Sapiens enzymology still remains to be elucidated.
CS2 exposure can give symptoms of Schizophrenia. (1) People with Schizophrenia have CS2 in their breath (2) other persons usually don't. Persons with Schizophrenia appears to react differently and have different partial arterial pressure on CO2 than others (3), (4). The very ground-level carbon to carbon

transfer used in CO2 fixation is done by constructions relying on the CODH/ ACS. The distribution of cerebral blood flow is highly sensitive to changes in the arterial CO2. (5) The CO2 regulations during sleep might be more vulnerable. (6) When the CS2 reactivity where measured between groups exposed to CS2 and not exposed to CS2, a remarkable difference was noted in the reactivity to CO2. (7) It has been shown in rats that low PaCO2 inhibits NMDA receptors. (8) The Thermodynamic backbone of human Neural transmittance is dependent on lipid and glucose metabolism, both is altered by the gating in the CODH/ACS tunnel A. One study suggests that the effect of CS2 on learning and memory ability in rats is related to the activity of NOS and the expression of nNOS in the hippocampus. (10)

B-vitamin metabolism disturbance

Exposure to carbon bisulfide results in an increased turnover of the B-vitamin complex (16, 17, 18, 19, 20) CS2 induced elevated serum lipid levels and decreased cholesterol synthesis (on rat, 176 ppm, inh) are reportedly blocked by feeding nicotinic acid at a dose of 40 mg/kg/da (20) Nicotinic acid has protective effects against CS2 poisoning. CS2 reduces the levels of Nicotinic Acid. (21 22 23 24 25)

Chelating effects on various essential trace metals

Carbon disulfide reacts with the amino groups of amino acids and proteins to form thiocarbamate in blood and tissues (36) thiocarbamates, possessing sulfhydryl groups, may chelate polyvalent inorganic ions. CS2 reacts with endogenous amines to form dithiocarbamates.(26) which could be metabolized back to CS2. An implication is the formation of acid labile CS2 (AL CS2) that will continue to increase, even at steady-state concentrations of CS2, as long as free CS2 is available to the tissue and adequate amine substrates are available. (26) An additional important finding was the slow elimination of AL CS2, suggesting that AL CS2 may accumulate in the body after repeated exposure to CS2. (26)
Dithiocarbamates are capable of chelating several polyvalent inorganic ions such as copper and zinc, and thus may inactivate numerous enzymes in which these ions are essential for activity. (27) The hypothesis of a chelating effect has been supported by the results of some studies (28), Reports of increase in zinc and copper excretion in exposed rats, but also increased copper levels in peripheral nervous tissue of exposed rats (28) (29). Copper and zinc ions are essential for the prosthetic groups of many enzymes. The neurotoxic action of carbon disulfide and its interference with the activity of many enzymes could partly be explained by chelating effects. Zinc is required for the activity of enzymes such as lactic acid dehydrogenase a, carbonic anhydrase, glutamate dehydrogenase, and alcohol dehydrogenase. Copper, represents a cofactor of pyridoxol, a form of vitamin B6. Copper is required for the proper functioning of enzymes such as cytochrome c oxidase, the coenzyme A dehydrogenase system, eg. dopamine ß hydroxylase. The loss of copper from the spinal cord is accompanied by cellular damage, producing tissue degeneration. Disturbances of the central and peripheral nervous systems,
resulting from carbon disulfide exposure, could be connected with the loss of copper due to chelation and consequent inhibitory effects on enzyme systems (30) like Tyrosinase and the very core carbon to carbon transfer.
LOX, Lysyl oxidase is copperdependent. The LOX activity are essential for the mechanical stability of the fibers and other supramolecular assemblies formed by these proteins and the elasticity of elastin. Because collagens and elastin are important components of the extracellular matrix, abnormalities in their modification can be expected to affect many tissues, as seen in lathyrism, a connective tissue disorder caused by the administration of ß-aminopropionitrile, an irreversible inhibitor of lysyl oxidases. (31) Extracellular copper enzymes initiate the formation of the lysine and hydroxylysine derived crosslinks in collagens and lysine-derived crosslinks in elastin. (32) CS2-mediated protein cross-linking occurs in vivo through the generation of Lys-Lys thiourea and that diethyldithiocarbamate can, through in vivo release of CS2, produce the same cross-linking structure. This observation supports the utility of cross-linking of peripheral proteins as a specific dosimeter of internal exposure for CS2 and provides a biomechanistic explanation to account for the high-molecular-weight neurofilament protein species isolated from rats exposed to CS2 or N, N-diethyldithiocarbamate. (33) High levels of homocysteine will irreversibly inhibit LOX. (34) One study suggest that LDL downregulation of LOX could contribute to the endothelial dysfunction caused by hypercholesterolemia, thus contributing to atherosclerotic plaque formation. (35) It has been reported that the glucose and lipid metabolism is disturbed by carbon disulfide both in

experimental animals and in exposed workers, but not to conclusive. There is a large body of indirect information associating abnormal energy metabolism in peripheral neuropathies caused by CS2.

The energy depletion

The pathomorphology of CS2 neuropathy resembles much like other samples originating from an impaired energy metabolism. (37) A study in rats shows the oxidative effects of CS2 exposure, a marked increase in cerebral cortex hippocampus, spinal cord and serum. Reactive oxygen species, Malondialdehyd. Ca2+ and Calmodulin levels increased in in Cerebral Cortex, hippocampus and spinal cord. (38) Carbon disulfide is used in viscose rayon plants as a solvent in the spinning process. It is known to have central and peripheral neurotoxic effects, and among the pleiotrophic conditions it causes are atherosclerotic change, diabetes mellitus, and coronary heart disease (39-42). In previous studies, the radiologic findings of carbon disulfide poisoning were diffuse or focal brain atrophy, infarcts in the basal ganglia, subcortial white matter and gray matter, and central demyelination (43-47). A few case reports have described the computed tomographic (CT) (43-47) or magnetic resonance imaging (MRI) findings (46, 47) Finding of decrease in the GSH contents and GSH-Px, CAT activities in cerebral cortex, hippocampus, spinal cord and serum. The activities of T-AOC also decreased in all three nerve tissues and serum, as time went on and symptom developed. Furthermore, significant correlations between LPO and gait abnormality were observed as symptom developed. Oxidation stress also resulted in increased Ca (2+) concentrations and calmodulin (CaM) levels increases in cerebral cortex, hippocampus and spinal cord. (48) Carbon disulfide intoxication results in alternations of microtubule and microfilament expression, and the alternations might be related to its neurotoxicity. fast changes in beta-tubulin and beta-actin in rats
exposed to CS2 could indicate a rapid change in the cytoskeleton metabolism: The beta-tubulin mRNA increased 207% and beta-actin 94% which might give insights in the metabokinetic prosperities of CS2 on a cytoskeletal level. (49) Many electrophiles toxicants cause synaptic dysfunction by unknown mechanisms. It is recognized that synaptic activity is regulated by the redox state of certain cysteine sulfhydryl groups on proteins. Research indicates that thiolates are receptors for the endogenous nitric oxide (NO) pathway and that subsequent reversible S-nitrosylation finely regulates a broad spectrum of synaptic activities. Electrophilic neurotoxicants like CS2 might, according to a hypothesized mechanism (16) produce synaptic toxicity by modifying these thiols. SNAP-25, NMDA, GAP-43, Methionine adenosyl transferases, v-ATPase are thiol-regulated proteins and protein complexes targeted by NO which further might explains the action of CS2 toxicity. One study suggests that the effect of CS2 on learning and memory ability in rats is related to the activity of NOS and the expression of nNOS in the hippocampus. (17)

Cancer and p53

The p53 gene has been proposed as tumour suppressor and a candidate susceptibility gene in schizophrenia. (52) results of one study indicate that occupational exposure results in a significant increase in P53 CGT>CTT transversions. (53) identified occupational exposure in combination with smoking as a significant risk factor for the mutation. It was concluded that AS-PCR of the P53 273rd codon transversions is a suitable technique for studying the effects of occupational exposure to CS2. (53), (57).
The CAC-Hålet Swedish site ADHD och ADD.

The etiology of Schizophrenia could be linked to endogenous Carbon disulfide CS2 production. The manufacture and exposure to endogenous CS2, could probably act in months after months before showing some of the symptoms of high CS2 exposure (Hallucinations, Psychosis and personality changes)

Suggestions

To ensure a proper analysis of earlier undetectable biomarkers one might consider gas-analysis from skin and breath as an biomarker and emission source, since the CS2 otherwise could leave undetected through the biggest organ - the skin. I propose analysis of total air content in a closed chamber with

subjects naked to avoid contamination with techniques for gas detection (59), (60) to reveal the biometrics of human endogenous CS2 production. Which has not been done to my knowledge.
By practising these suggestions it might be easier to understand and prevent the disease by detecting the prodromal and active systems conditions besides the genetic vulnerability and map out what role CS2 play in the etiology of Schizophrenia.

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