To The Senate Select Committee
on... Mental Health Issues...
Parliament House Canberra...

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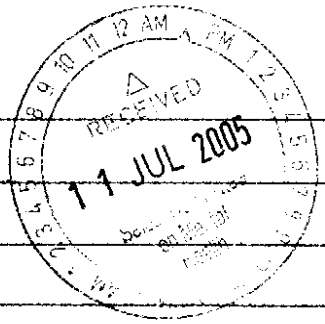

... July 6th 2005...

... To the Committee.. and Whom it may concern...

... Hello..

I only recently heard of this committee and its current inquiry... and concerns... So I hope I am not too late to voice some issues of relevance and very painful and disappointing personal experience...

... My submission to the Committee concerns the way 'Psychiatric Ideas of Disorders' can be overused and **misused** as discrimination and abuses of Social-Economic Power against vulnerable powerless groups of people... in particular in my case children and teenagers...

... Mothers-Fathers... Doctors-Nurses can, with too little accountability and often just mercenary convenience... use 'Ideas of Disorders'... to traduce and trespass upon young people trying to physically-mentally-emotionally-philosophically etc... mature bigger.. more active.. different and independent lives beyond childhood introductions that the relevant adults may have hitherto provided and be accustomed to...

... 'Ideas of Disorders' can be too readily sought and factitiously provided to prevent and misunderstand... young people beginning and fulfilling these personal-social--personality maturations... that allow them to separate and differentiate independent lives and minds away from parent's childhood expectations and experience of them...

... Young developing people who cannot adequately protect themselves from this can be left with enduring.. disabling.. unresolved Iatrogenic Psychiatric Damage...

... Psychiatric Ideas of Disorders used traducingly against them... can leave people Nihilistically bastardised out of developing and living any sort of physically--mentally active life beyond childhood... Because How can you live when every physical-mental activity and arousal of your life is Psychiatrically bastardised as a Disorder...?...

...As written and emphasised by some parts of the Psychiatric Profession...eg. Thomas Szasz...
...'Ideas of Psychiatric Disorders' can be overused and misused as abuses of Power...
and expressions of Ignorance...Intolerance for Diversity of Life and living practises...
Sometimes Ideas of Disorders are not about Medical Care... they are mercenary conveniences
and abuses of Relationship and Social Power... trespassing upon undergated Civil and Human Rights
- Liberty, Security, Self-Determination of Person...
- Freedom of Movement, Freedom of Expression... For example...
...Frequently abrogating problems in living environments... Inadequate housing.. food.. clothing..
heating.. exercise.. other living facilities and a persons lack of Social-Economic and
Relationship Power... with which to assertively act and protect physical and mental wellbeing...

...I would expect that the Committee has more than enough documentation of personal
stories as part of its Inquiry's evidence and submission... However, I attach my
disappointing and destructive experience as evidence of...

"The Iatrogenic Suffering that can be inflicted by physicians who act on
the basis of inadequate information and false assumptions..."

...Accountability and Self-Regulation in the Medical-Nursing Professions doesn't really
work as a private Medical Concern...
Whilst the Medical Profession protest to the Federal Government about Immigration Detention
Centres and false imprisonment... solitary confinement... especially of Women and Children
and how Physically and Mentally destructive this can be... The Medical Profession needs
to clean up its own backyard... of false imprisonments and solitary confinement of children
- legal minors... Trespassing - Negligent Grimm Fairy Tales of Hansel and Gretel ChildCare...

Yours Sincerely.. detach...

'Textbook of Biochemistry with Clinical Correlations 3rd Ed. T.S. Devlin...

CLIN. CORR. 12.6
DISORDERS OF TYROSINE METABOLISM

TYROSINEMIAS

The absence or deficiency of cytosolic tyrosine transaminase (tyrosine aminotransferase, TAT) is responsible for the accumulation and excretion of tyrosine and metabolites including *N*-acetyltyrosine, *p*-hydroxyphenylpyruvate, *p*-hydroxyphenyllactate, *p*-hydroxyphenylacetate, and tyramine. Since *p*-hydroxyphenylpyruvate, the presumed precursor of some of the other products, is also the product of the transaminase, it is likely that these products come from mitochondrial transaminases or oxidases in extrahepatic tissues. The disease is characterized by eye and skin lesions, and most but not all of the cases reported have been mentally retarded. This condition is called oculocutaneous or type II tyrosinemia.

Type I, hepatorenal tyrosinemia, is a more serious disease involving liver failure, renal tubular dysfunction, rickets, and polyneuropathy in addition to excretion of tyrosine, other amino acids, and other metabolites. All of this appears to be caused by a deficiency of fumarylacetoacetate hydrolase. For unknown reasons there is an increased risk of hepatoma development in children with tyrosinemia.

Deficiency of *p*-hydroxylphenylpyruvate oxidase is believed to be responsible for neonatal tyrosinemia, which is usually a temporary condition and in some cases responds to ascorbic acid, given on the hypothesis that this compound protects the enzyme from substrate inhibition.

The very different consequences of deficiencies at various points in tyrosine metabolism show the necessity of analyzing all of the factors that might be relevant and avoiding a simplistic explanation. Interruption of the pathway at homogentisate oxidation causes this compound to accumulate without any other metabolic effects. Homogentisate is not an intrinsically reactive compound, and it does not alter any enzyme before it is excreted. In contrast, in the absence of its hydrolase, fumarylacetoacetate accumulation causes maleylacetoacetate to accumulate as well and this is chemically reactive, especially combining with sulfhydryl compounds. Another toxic compound, succinylacetone, has been suggested as a secondary product of fumarylacetoacetate metabolism that might be responsible for some of the biochemical lesions in this disease.

ALBINISM

Skin and hair color are controlled by an unknown number of genetic loci in humans; in mice 147 genes have been identified in color determination. It is not surprising, therefore, that skin color exists in infinite variations and also that formation of pigment can be interfered with in many ways. Many conditions have been described in which the skin has little or no pigment, but the chemical basis is not established for any except classical albinism. In this condition the enzyme tyrosinase is deficient and melanin is not formed. Lack of pigment in the skin makes albinos sensitive to sunlight, which may cause carcinoma of the skin in addition to burns; lack of pigment in the eyes causes photophobia. Lack of eye pigment does not imply impaired eyesight; a description of albinos among American Indians in 1699 indicated that their vision at night was superior to normal.

ALCAPTONURIA

The first condition to be identified as an "inborn error of metabolism" was alcaptonuria. People deficient in homogentisate oxidase excrete almost all ingested tyrosine as homogentisic acid in their urine. This hydroquinone is colorless, but on standing it autooxidizes to the corresponding quinone, which polymerizes to form an intensely dark color. Concern about the dark urine is the only consequence of this condition early in life. Homogentisate is slowly oxidized to pigments that are deposited in bones, connective tissue, and other organs. This generalized pigmentation is called ochronosis because of the ochre color seen in the light microscope. Pigment deposition is thought to be responsible for the arthritis that develops in many alcaptonuric individuals, especially in males.

The analysis of alcaptonuria by Archibald Garrod who first indicated its genetic basis as an autosomal recessive deficiency condition includes an unusual historical description of the condition. This is of great value in appreciating the iatrogenic suffering that can be inflicted by physicians who act on the basis of inadequate information and false assumptions.

Fellman, J. H., Varbellinghen, P. J., Jones, R. T. and Koler, R. D. Soluble and mitochondrial tyrosine aminotransferase. Relationship to human tyrosinuria. *Biochemistry* 8:615, 1969; and Kvittingen, E. A. Hereditary tyrosinemia type I.—An overview. *Scand. J. Clin. Lab. Invest.* 46:27, 1986.

'This is of great value in appreciating the iatrogenic suffering that can be inflicted by physicians who act on the basis of inadequate information and false assumptions...'

atom of the side chain by a copper-containing enzyme located in chromaffin granules. Some of the product, *norepinephrine*, is stored in secretory granules until the cells are stimulated to release the hormone into the blood. The larger part of the norepinephrine is methylated by a relatively nonspecific **phenylethanolamine *N*-methyltransferase** to become *epinephrine*, which is also stored in the chromaffin granules. Epinephrine and norepinephrine are hormones known collectively as catecholamines. Norepinephrine is stored in vesicles in the termini of axons to be released for synaptic transmission of nerve impulses. They affect the physiological functions and metabolism of most organs very rapidly, at least in some cases by stimulating the synthesis of cAMP.