EXHIBIT CE

Ms J. Culver,
Office of the DPP,
Castlereagh St,
SYDNEY NSW 2000

19 March 2003

Dear Ms Culver,

Re: R-v-FOLBIGG
IL-10 GENE THEORY

I refer to the above matter and acknowledge receipt of your 2 page letter dated 17 March 2003, in which certain questions have been asked of me in relation to Dr Drucker's paper and the IL-10 gene. I have read "Association of IL-10 Genotype with Sudden Infant Death Syndrome", Human Immunology 61, 1270 1273 (2000) in its entirety.

I set out my responses to these questions below:

"1. Briefly, what is the IL-10 gene theory in respect to SIDS?"

This is a theory (and therefore unproven) which attempts to explain the Sudden Infant Death Syndrome ("SIDS"). SIDS is a term which was invented years ago to assist in explaining the deaths of certain infants who died suddenly and unexpectedly, and who had been previously generally well. These infants were aged between about 3-6 months but were not older than 12 months. SIDS replaces the cause of death "Undetermined" and so usually spares the parents a detailed police investigation and later inquest.

The IL-10 gene theory has as its core the proposition that some SIDS infants have the IL-10 gene, especially the Il-10-592*A genotype in greater predominance than other infants ("controls"). This gene has a normal function by regulating IL-10 (interleukin 10), a natural anti-inflammatory chemical in the body which suppresses inflammation and promotes antibody production. If an infant has this gene, it is theorized, that infant is at risk of developing an overwhelming infection at around the age of 4 months, when its own immune system is still in an immature state. If this infant has the gene, there may be an imbalance of chemicals in the body such that the infant is more likely to develop an overwhelming infection and then die suddenly.

If this is true, one would expect that SIDS would run in families, given its genetic aetiology, whereas this is not the case. SIDS is sporadic, and is not a genetically inherited condition.

2.***Is this theory accepted in the medical community generally?***

I do not accept this theory and neither does the forensic community or wider medical community. This theory and other similar ones have been published and presented at

conferences over the past 6-7 years to explain some SIDS deaths, but in fact there has not been an enthusiastic response by the medical community to broadly accept this theory.

3.Has the theory been subjected to testing or review independently of the studies conducted by/through Dr Drucker?

I am not aware of any studies which have reviewed this theory and independently evaluated it. In this paper, only 23 cases of so-called SIDS were tested for this gene. This is a very small group and caution should be exercised in drawing conclusions based on such a small study. I would also like to evaluate each case myself rather than rely on another pathologist's diagnosis of SIDS.

4.Is there a proper or reliable scientific basis for drawing an association between the A-allele of the IL-10 gene and SIDS?

I do not believe so. When closely examined, this paper is confusing and contradictory. On the one hand, Dr Drucker states SIDS infants carry this gene in greater percentages compared with normal infants. But on the other hand, he states:

"a deficit in IL-10 may contribute in two ways to SIDS, that is, by a tardy initiation of protective antibody production in the infant and a lower capacity to inhibit inflammatory cytokine production."

Clearly, however, the gene exists and has a function in life. That its presence can somehow explain the deaths of these infants is in my view unscientific and implausible.

5.Were the tests performed by Dr Drucker in this matter performed in a reliable, scientific and repeatable manner?

In all likelihood, yes, although the small numbers involved in the study make the conclusions invalid.

6.Can those test results be regarded as accurate and reliable given that the DNA was extracted from histological samples in paraffin blocks and that, according to Dr Drucker, multiple repeats in the analysis of that material had to be made due to "the very degraded nature of the DNA"?

I would be cautious in relation to the interpretation of those results due to this factor. Although the preservation of the specimens in paraffin wax would be reasonable, nevertheless a 10 year period between the child's death and later testing may lead to unreliable results. I am unaware of any archived material being used in this way at the moment. Degraded DNA may make these results unreliable and invalid.

7.Is there anything known professionally about Dr Drucker which may undermine or discredit his authority in reaching the results and conclusions which he purports to reach?

Not that I am aware of, however it may be fair to say that he is probably a full-time academic microbiological researcher, and may have had very little to no direct experience in investigating SIDS deaths. Whilst I have no view in relation to his other work, the other articles referred to in the article are also of an infectious/microbiological nature.

Researchers of necessity develop their research around an area which naturally interests them. They often then become expert in that one area, and tend to theorise based on their area of expertise. Extrapolating from that area of expertise into an unrelated area may be dangerous especially in a medico-legal setting.

There is no broad acceptance that SIDS is infectious in origin. At this stage, I reject this particular theory as having any role in the aetiology of SIDS.

Yours,

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