Advances in Neuroimmune Biology 3 (2012) 229–234
DOI 10.3233/NIB-012918
IOS Press

Introduction

Stress and My Life

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INTRODUCTION

Hans Selye, the father of stress concept has never really defined stress. The best he could do was to say that stress induced a *general adaptation syndrome*. Adapted animals resisted stress [1]. However, stress also had to do with disease and this was the aspect which Selye emphasized since 1955, when he published in Science the article *Stress and disease* [2]. Apparently if stress was causing disease it was highly fundable by granting agencies. So I herd about this aspect of stress only in the Selye Institute and this has been transmitted to the public conscience, as well. Nowadays people are "stressed out" when things are going really bad.

Selye saw the thymus shrink, but in those days he had no knowledge of what the thymus was doing. It was not known either what the spleen was doing or that the pituitary gland was connected to the hypothalamus. Despite of these difficulties he was very sure of the significance of his findings on stress.

I was in his laboratory in 1966–67. By that time I was an Immunologist and prior to coming to Canada, I worked as a Research Fellow at the Hungarian Academy of Sciences. The function of the thymus has been just elucidated a few years earlier by Miller in 1962 [3]. It was shown to be a central immune organ of generating thymus derived (T) lymphocytes. T lymphocytes were characterized as important in immunoregulation and in cell mediated and humoral immunity. That the spleen contains antibody producing cells was discovered by Gowans and McGregor in 1964 [4]. The linkage of the pituitary gland to the hypothatamus has been discovered during the time I was in the institute. So Selye was much ahead of his time in 1936, when he discovered the Stress syndrome. He discovered something he did not fully understand. The relevant information about the HPA axis and the immune system was not available to him. Selye was not aware of what the thymus or spleen were doing even when I came to his laboratory.

I told him one day that the observation that stress shrinks the thymus and lymphoid organs, testifies for the existence of *neuroimmune interaction. But what the CNS would have to do with the immune system?* Nobody knew the answer to this question in those days.

Selye was interested for a while in this problem, but eventually he forgot about our conversation. So I decided to answer the question *what is the significance of CNS – immune interaction?* I was ready to dedicate my life time research for answering this question, if it was necessary. Well it took 30 years to answer the question. But the project was not finished with this, this was only the beginning of figuring out how this Neuroimmune Supersystem works, which has to do with everything, in health and disease and from conception till death. Below I give a short outline of my research career in relation to stress.

After I left the Selye Institute in 1967 I made the decision to get my PHD in Tumor Immunology with Alec Sehon, who was to move from McGill University in Montreal to Winnipeg in order to set up the first Department of Immunology in Canada. I finished my PhD in Tumor Immunology in 1972, and Sehon invited me to become a Staff Member in the Department of Immunology. So I made the decision to stay in Canada.

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ISSN 1878-948X/12/$27.50

The opportunity came to investigate what the CNS has to do with immune function, when Dr Eva Nagy came to my laboratory from the University of Szeged, Hungary in 1976. She was an Assistant Professor of Endocrinology and an expert on hypohysectomy, which is a very delicate operation.

I asked Eva to apply for an MRC scholarship and she got one. This was for 3 years. So we started to do hypohysectomies on rats and saw deficiency of adaptive immune responses, which was published in 1978 [3]. This was the first definite indication that *pituitary gland is a major immunoregulator*.

In those days in the literature there was Selye's discovery that the involution of the thymus and lymphoid tissue was caused by arenal glucocticoids [4] and Ader and Cohen described Pavlovian conditioning of the immune rections [5], which is indirect evidence for neuroimmune interaction and even today its mechanisms are not completely clear. In 1991 Besedovsky and colleages championed that the brain and immune system interacts and described a substance mediating this interaction, which later on turned out to be inteleukin 1 (IL 1) [6]. For more information on the role of the HPA axis, please see the related chapter in this book [7].

In subsequent experiments we showed that the adaptive immune system (eg. *humoral and cell mediated immunity*) is maintained by growth and lactogenic hormones (GLH), which involve growth hormone (GH), prolactin (PRL) and placental lactogens (PL). *Adrenocoticotropic hormone* (ACTH) inhibited immune function in these experiments [8, 9]. This was true after using replacement (physiological) does of hormones. *Contact hypersensitivity reactions*, which is a form of cell mediated immunity, designated as delayed – type hypersensitivity, was also pituitary dependent [10]. *Adjuvant arthritis* also depended for development on GH or PRL [11]. The dopamine agonist drug, *bromocriptine*, was as immunosuppressive as much as was HYPOX [11]. This drug inhibits PRL secretion.

In 1979 Eva's fellowship was over and I re-applied to the Medical Research Council of Canada and funding was denied. The answer from my Immunologist colleagues was: "we cannot fund this because this is not Immunology". Well I tried a number of other funding councils but never-ever gut funded by MRC nor by MHRC (Medical and Health Research Council of Canada) which replaced MRC. This created immense difficulties for my laboratory for the rest of my career (1979–2009). The Immunology committee managed to kill Neurimmune Biology (NIB) in Canada. NIB did not keep up in Canada with the development of this science word wide. What should have been done is to establish a separate granting council for NIB. This was a highly original and novel area of research, where there were a lot of unknown questions and lack of details. Such applications do not stand up well against the scrutiny and competitiveness of standard grant committees. But a special arrangement was never done for NIB and to the best of my knowledge, there is no research on this subject in Canada even today.

How did we survive with Eva? With tremendous difficulties. There was no other source of significant funding in Canada. My Dean, Nicholas Anthonisen, who was a distinguished scientist in his own right, recognized our achievements and provided money for computers and for equipment. We collaborated with people who had money. Prof. Henry Friesen, Drs. Edris Sabbini, Edward Baral, were our long time collaborators. Collaboration required the identification of projects, which interested both sides. So we worked on molecular biology of hormonal immunoregulation, on the submandibular gland and on tumor immunology. The Thorlakson foundation in Winnipeg helped me with a little grunt. Occasionally I got little money from other small foundations. I wrote a grant to NIH and my project was stolen. Funding was denied. So, welcome to the competitive world of science! This is how science policy almost destroyed my research for a lifetime.

I found out that I am able to do scientific books, so I done a total of 15 books to date. Frequently I also wrote review papers. No grants were required for doing books and review papers and this activity registered in my curriculum. Dr Anthonisen once remarked that "nobody can produce results than I do".

My first book, *Pituitary function and Immunity* was published in 1986 [14]. This volume summarized the knowledge on the subject matter up to date.

For the past 10 years or so I collaborated with my colleages in the University of Aguascalientes in Mexico. Here we work on the hypothalamus and pituiry gland in relation to immunoregulation. The results we are getting are very interesting and important. So, let us get back to science again, which I love so much.

In Dr. Friesen's laboratory I spent my sabbatical year in 1990 and worked on the molecular biology of NIB. We observed that the gene c-myc and DNA and RNA synthesis was regulated by GH and PRL in spleen and thymus of rats. Both C-myc and nucleic acid synthesis indicated cell activation, proliferation. DNA synthesis in the spleen and thymus directly correlated with the grade of inflammatory response in skin (cell mediated

immunity to dinitrochlorobenzene, DNCB) [15]. We also observed that ovine GH and PRL induced insulin like growth factor-I (IGF-I) synthesis in thymus of HYPOX rats [16]. This answered the question if IGF-I played a role in lymphocyte proliferation induced by GH and PRL.

We had an opportunity to study prolactin bioactivity in sera of patents with rheumatoid arthritis. We found that by radioimmunoassay PRL was normal, but bioactivity was decreased when measured with the Nb2 lymphoma bioassay [17]. This observation has been confirmed in the recent literature [18, 19]

Children with malformation of the pituitary gland do not survive. This observation suggests that rats would die after complete HYPOX. However, long surviving HYPOX animals may live to 6–8 months. So we took surviving HYOX rats and measured serum PRL levels. Initially the level amounted to a few nanogramms, but serum PRL rose gradually up to 40% of normal levels by day 40 after HYPOX. Hematologic parameters and immune function was normal in such animals. However, when we neutralized the residual PRL in the blood of such surviving animals with antibodies, they lost weight, became quickly and severely anemic and immunodeficient and died within 14 days [20]. These experiments indicate that PRL is capable of maintaining vital bodily functions for up to 8 months.

We showed that GLH regulated the bone marrow [21, 22]. Some old papers were available on this subject but the regulation of bone marrow function by pituitary hormones was not appreciated in modern times. It is interesting that the bone marrow does not regress in Acute Phase Reactions (APR) but the thymus regresses. The bone marrow is activated during APR. Some cytokines of APR, such as IL6 and GMCSF may activate the BM during acute illness.

During our studies in cancer immunology we discovered that the anti estrogenic agents had immunoregulatory effects; In those days anti estrogenic drugs (eg. tamoxifen, toremifen) were used to treat estrogen receptor positive tumors. Nothing was known about the immunological effect of these drugs. We showed for the first time that these drugs sensitize tumor cells for killer cell mediated lyses and at the same time stimulated also the cytolytic effects of killer T and natural killer (NK) cells. These results suggested that it would be advantageous if we treat tumor bearing animals with both killer cells and anti estrogenic drugs. We did such experiments in animals with excellent results. The majority of the animals were cured of lethal cancer. We called this approach *combination immunotherapy of cancer* [23–30]. Today combination immunotherapy is used for the treatment of patients with good results.

Book: Berczi I, Szelenyi J, Editors, Advances in *Psychoneuroimmunology*. Plenum Press, New York, 1994. [31]. This book contained the papers of a meeting in Budapest, which commemorated Hungarian Scientists (Hans Selye, Jancsó Miklós, Andor Szentivanyi) who contributed significantly to the development of NIB.

We adrenalectomized (ADX) mice and found that ADX mice showed an increased sensitivity (∼500 times) to lipid A, which is the toxic moiety of LPS endotoxin. When the ADX mice received replacement doses of the glucorticoid agonist drug, dexamethason, the animals survived after endotoxin challenge, similarly to normal controls [32]. This paper testifies that the HPA axis mediates a rapid host defense against toxic insults by secreting glucocrticoids. The significance of the neuroimmune host defense system was discussed in a review paper [33].

This was the time for answering the question *what the brain has to do with the immune system?* The Brain, the Endocrine and the Immune Systems form a systemic regulatory circuitry, which regulates everything in higher organisms. The stress reaction is analogous to the acute phase response (APR), this response is the ultimate attempt by the neuroimmune system to defend the host against life threatening insults. During APR the entire organism participates in host defense. Innate immune-neural-endocrine-metabolic-, leukocyte and acute phase protein responses are activated. APR, or febrile illness occurs frequently in our lifetime, and in most instances we survive. So APR is a very effective defense response against dangerous insults [34].

Dr. Sabbadini worked with an immunosuppresor enzyme from the submandibular gland of mice. It was found to be *glandular kallikrein* and it could suppress immune reactions in animals. We studied the hormones what regulated this system. Prolactin, sex steroid hormones and thyroid hormone showed a regulatory influence. Glandular kallikrein (GK) is major immunoregulatory enzyme in the saliva and the gut and is also in the blood stream [35, 36]. We also studied the commercial potential of GK. We found that it suppressed the generation of oral immunological tolerance in rats. GK can be used for the suppression of autoimmune reactions in animals and in man and it is being commercialized for this purpose.

I wrote a theoretical review paper with my colleagues. The subject was hormonal regulation of self tolerance and autoimmune disease [33]. By this time it was clear that immune function is regulated by hormones. Normally the immune system shows self

tolerance and reacts to "foreign" antigens. Autoimmune reactions are exception to the rule. So the question was asked: do endocrine abnormalities lead to abnormal immune function? Some evidence for rheumatoid arthritis suggested a positive answer to this question.

In another review paper we discuss the thymus response to APR. Acute involution occurs in the thymus in response to the increased level of glucocorticoids and of the emergence of suppressor regulatory T cells (the information on Ts/r is more recent). The thymus is suppressed along with the adaptive immune system, which is rendered inactive during APR. The innate immune system becomes forcefully activated during APR [38].

We also discussed with Lorand Bertok, and Donna Chow, the role of natural immunity in neuroimmune host defense. We describe that during APR it is the innate immune system which gets activated and would defend the host against life threatening insults. The innate immune system is capable of instantaneous response, whereas the adaptive immune system needs 7–10 days for a response. This system is useless for acute responses [39].

It was my desire to fund a review journal which will present the research findings in Neuroimmune Biology. I created this term for the designation of the field that deals with neroimmune interaction. I call it Biology because of the interdisciplinary nature of this science. Biology is the proper term to describe it. My co-Editor, Dr Andor Szentivanyi blessed this term, so we call the book series published by Elsevier, Neuroimmune Biology Berczi I, Szentivanyi A, Series Editors. *Neuroimmune Biology*. A serial publication of books initiated in 2001. Elsevier Publisher, Amsterdam [40]. (www.elsevier.com/locate/nib).

Nine volumes have been printed; *Volume 1; New Foundation of Biology* [41];

Vol 2: *Growth and lactgenic hormones* [42]*; Vol. 3 The Immune-Neuroendocrine Circuitry. History and Progress* [43]. *Vol. 4. The Neuroendocrine Immune Network in Ageing* [44]. *Vol. 5. Natural Immunity* [45]. *Vol. 6. Cytokines and the Brain* [46]. *Vol. 7. The Hypothalamus-Pituitary-Adrenal Axis* [47]. *Vol. 8. Neurogenic inflammation in health and disease* [48]. *Vol. 9. The brain and host defense* [49].

Elsevier stopped printing this book series in 2009. The justification was that they did not make enough money. I wanted a journal in the first time. Elsevier had a number of immunology journals. So they gave me a book series. I noticed very early that they did not promote properly these books. It was difficult to find the website if you wanted to order. We were working on volume 8 when they told me that they will stop producing, they did not make enough money they told me. Gush! I provided the manuscripts in camera ready format so producing a book cost them below $ 10. They sold it per copy at about $ 175. They could print exactly the copies they sold. On each copy they would make say $ 150, taking off some money for handling expenses. This comes to some 600% profit we are talking about. Rather, the established journals made even more profits, so Elsevier wanted to cut down the competition by this move. This is what I believe. They took away 10 years from my creative life, and the series I started was killed. Asi es la vida, we say in Mexico.

In 2011 I had the opportunity to start a web-based review journal, *Advances in Neurimmune Biology*, Editors of Chief: Istvan Berczi and Tosihiko Katafuchi. It is volume 4 we are working on now. This paper will appear in volume 4. *Stress and immunity* [50].

In Canada I had to retire in 2009. Since 2011 I am an Invited Professor at the University of Aguascalientes, Agu., Mexico. I was just given a staff appointment with the prospects of progressing in my career. I am very satisfied to have this opportunity in my golden years. We work on the hypothalamus and the pituitary gland and immune function. Our research findings are summarized in the chapter: Vasopressin, oxytocin and immune function printed in this book [51]. I is a pleasure of living in Mexico, which is multiplied by my lovely, intelligent and very creative wife, Maru Quintanar.

Three other papers which are in this volume discuss important current issues in NIB, such as Immunocompetence, adaptive and innate immunity in acute illness, corticotropin releasing hormone and immune function [52–54].

I wrote 3 theoretical papers recently: one is about immunoconversion in the acute phase response. During APR adaptive immune function is suppressed and innate immunity is amplified. Therefore the neuroimmune regulatory network converts from homeostatic (adaptive – innate) regulation to solely innate regulation, which is designated as *immunoconversion* [55]. I discuss in the next paper that the 3 systems, the Nervous- Endocrine- and Immune Systems form the regulatory circuitry, which regulates the entire organism. Because this regulatory unit is made of more than one system I coined the name, Neuroimmune Supersystem [56]. With my colleagues I discuss neuroimmune regulation in immunocompetence, acute illness, and healing. Briefly in acute illness the CRH-Pituitary-adrenal-Innate immune axis is activated. Vasopressin supports CRH at this stage.

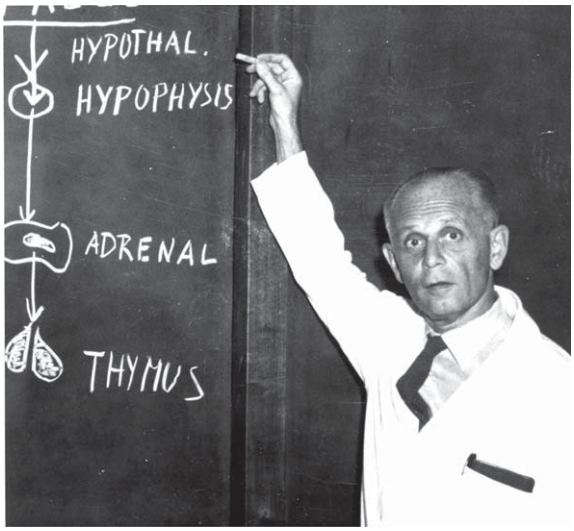

Fig. 1. Selye illustrates his discovery.

However, during the chronic phase of the disease CRH will subside and VP becomes the hypothalamic regulator. VP leads the organism to healing and recovery, as it regulates both the HPA axis and PRL [57].

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