J M Lundberg group at Karolinska Institute

I) How it started

I was introduced to medical science at the age 10 by my uncle Dag Lundberg who took a blood sample from my finger, stained a slide and let me look in a microscope on my blood cells, the" red ones helping me breathing and the white ones helping me to stay healthy and fight infections". The cells looked so beautiful and mysterious and stayed as an unforgettable memory. The next step happened at my scholarship experience after student examen at Gustavus Adolphus college, (GAC) Minnesota, USA. I had an icehockey scholarship (and funds from the Kings foundation) to pay for a year studying chemistry and psychology. Unfortunately, I got injured early in the season, so I had a lot of time for studies, which was not so common among the athletes. Then I learned about two Swedish scientists: A Dahlstrom and Kjell Fuxe who had mapped out monoamine containing neuronal systems in the brain that specifically were characterized by noradrenaline, dopamine and serotonin. Increasing evidence suggested that these neurons could have a role in a variety of brain functions like alertness, mood and movement control. Since GAC had a Swedish heritage and I was Swedish, this Swedish science origin discussion became very relevant to me.

After returning to Sweden and starting at Gothenburg medical school in 1973, I trained for a while in Annica Dahlstrom's lab and learned more about the peripheral nervous system. Furthermore, the courses in Pharmacology and Physiology had excellent teachers like Prof Arvid Carlsson and Prof Björn Folkow lecturing about neuropsychopharmacology and cardiovascular autonomic nervous control, respectively. I became increasingly interested in the mechanisms of chemical nervous system transmission.

II) Discovery of vasoactive intestinal polypeptide (VIP) in cholinergic neurons of exocrine glands

The career changing seminar I attended in fall of 1977 was delivered by Dr Tomas Hökfelt from the Karolinska Institutet (KI), who talked about peptides as mediators of nerve signaling, more specifically substance P(SP), which had been discovered by another Swedish KI scientist (Ulf von Euler). Elegant immunohistochemistry demonstrated the presence of SP in small sensory spinal neurons with central branches into dorsal horn of spinal cord and peripheral ramifications into eg. the skin. This was a novel new field opening up to be studied in more detail about further localization and especially potential functional and pharmacological applications. I had been considering taking a pause in my medical studies to do a PhD thesis (doktorsavhandling) and now this interesting neuropeptide area came in front of my eyes. In addition, the education government decided that top grades (which I had achieved in all subjects) was not as important for medical studies so grades were removed from exams. Consequently, future allocation of medical internships should not be based on grades in courses but on a lottery procedure. Hence, this situation (not being able to "influence "your future) further triggered my desire to add on

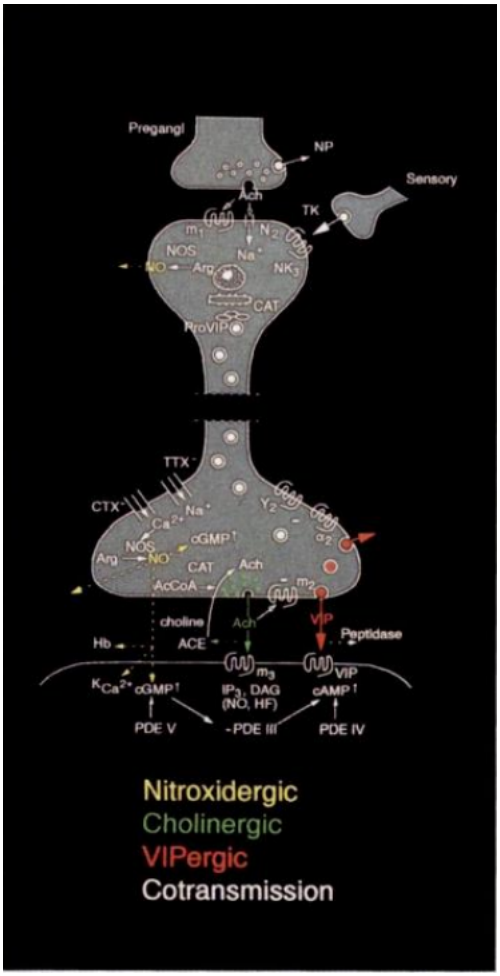
further merits (a PhD) to my CV and to contact Dr Hökfelt and ask if I could come to his lab and learn some new techniques? His lab was already quite full of PhD students and guest scientists, so I had to prove myself to be worthy of a new specific task. Here my new knowledge about peripheral nerves and ganglia came handy. After a test period I was allowed to be a PhD student (without any position or pay) and start to study the peripheral nerve distribution of various neuropeptides (Vasoactive intestinal polypeptide, VIP discovered by V Mutt at KI) and enkephalins, in addition to SP.
At the same time, I wanted to get more functional aspects of peripheral nerves into my thesis program. I therefore went to the Pharmacology department where prof Sune Rosell had interests in pharmacological neuropeptide (SP) antagonists and access to various large animal models. After talking to some science groups there, I finally agreed to work with an experimental clinician Doc Anders Änggård from the ear, nose and throat (ENT) department at the Karolinska hospital. He was an expert in cat microsurgery, autonomic nerve stimulations and blood flow determinations. I now needed to decide a model system for my functional peptide transmission studies.

A fundamental discovery happened in 1979; lumbar sympathetic ganglia in the cat contains classically cholinergic (acetylcholinesterase positive) neurons innervating skin sweat glands of hind paws via the sciatic nerve. We now detected VIP immunoreactivity (IR) in the same neurons, illustrating coexistence of two potentially active chemical messengers VIP and Acetylcholine (ACh). But how could we study the transmitter mechanisms, like mediator release and effects on blood flow and exocrine secretion in a simpler, more easily accessible system? We chose the cat submandibular gland where the same coexistence seemed to exist between acetylcholine and VIP. Further, there was a classic atropine-resistant noncholinergic vasodilatation upon stimulation of the parasympathetic nerves suggesting presence of additional mediator than ACh. In the thesis plan, the following aspects of Dales transmitter criteria were studied:

1) How widespread was the coexistence of ACh and VIP -IR in neurons of exocrine glands?
2) What was the subcellular storage of ACh and VIP in neurotransmitter storage granules?
3) How was the supply of VIP to nerve endings from cell body synthesis?
4) How was release of VIP and ACh upon parasympathetic stimulation with different frequencies?
5) What were the functional responses to VIP or ACh and/or combinations thereof?
6) Could the nerve stimulation effect on salivary secretion or increase in blood flow be modified by atropine or neutralizing VIP antisera?

These aspects were defended in my thesis in 1981 (Fig 1).

Figure 1 VIPergic Cotransmission. From *Lundberg Pharmacology Review 1996*



VIP and Ach had different subcellular storage and supply. VIP being preferentially stored in large dense cored vesicles, synthesized in the cell body and supplied to terminals by axonal transport. VIP and Ach seemed to have complimentary roles on blood flow and salivary secretion, whereby VIP was primarily a vasodilator, released at high stimulation frequencies, also enhancing cholinergic salivation.

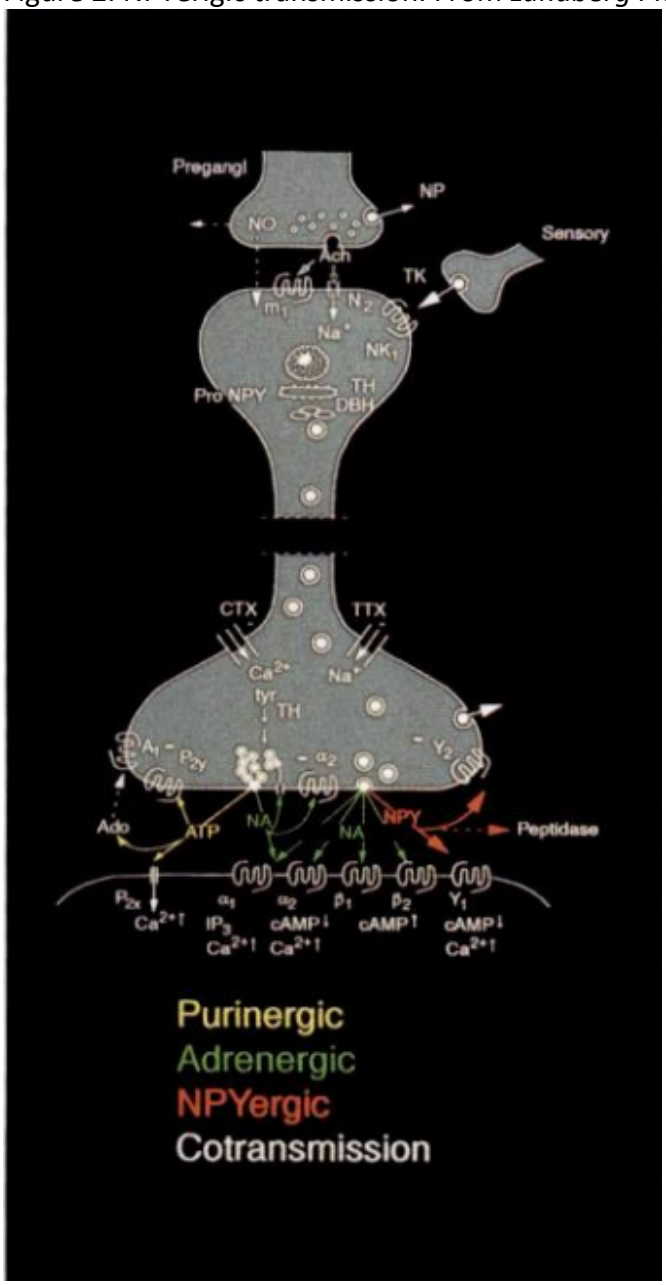
Other labs outside my two thesis tutors also contributed to these activities: Prof V Mutt (VIP peptide supply) Dr J Fahrenkrug (VIP measures, VIP antisera), Prof S Brimijoin (axonal transport).

III) Neuropeptide Y in sympathetic neurons-stress release

Due to a vast collaboration network in Dr Hökfelt's lab an antiserum to avian pancreatic polypeptide (APP) was tested from Dr Kimmel. This antibody unexpectedly stained noradrenergic (NA) sympathetic neurons. But what was the nature of this immunoreactivity (IR)? The potential solution came later when Dr K Tatemoto in Prof Viktor Mutts lab first isolated another member of the pancreatic polypeptide family (PYY); peptide (P) with N and C terminal tyrosine (Y) from

pig intestine. This peptide had very potent vasoconstrictor activities and inhibited intestinal motility being present in colonic endocrine cells. Soon thereafter a similar peptide; neuropeptide Y (NPY) was isolated from pig brain. We found similar staining as APP-IR of sympathetic nerves with NPY antiserum having a wide distribution in peripheral tissues including heart, vascular system and vas deferens. Interestingly, NPY peptide blocked the APP-IR staining suggesting that the APP antiserum cross reacted with NPY in the tissue. Hence, it was logical to compare the biological activity of NPY. NPY like PYY was a potent vasoconstrictor and in addition inhibited the biphasic contractions of vas deferens induced by electrical stimulation suggesting prejunctional activity on NA and adenosine triphosphate (ATP) transmitter release (Fig 2).

Figure 2. NPYerigic transmission. From *Lundberg Pharmacology Review 1996.*

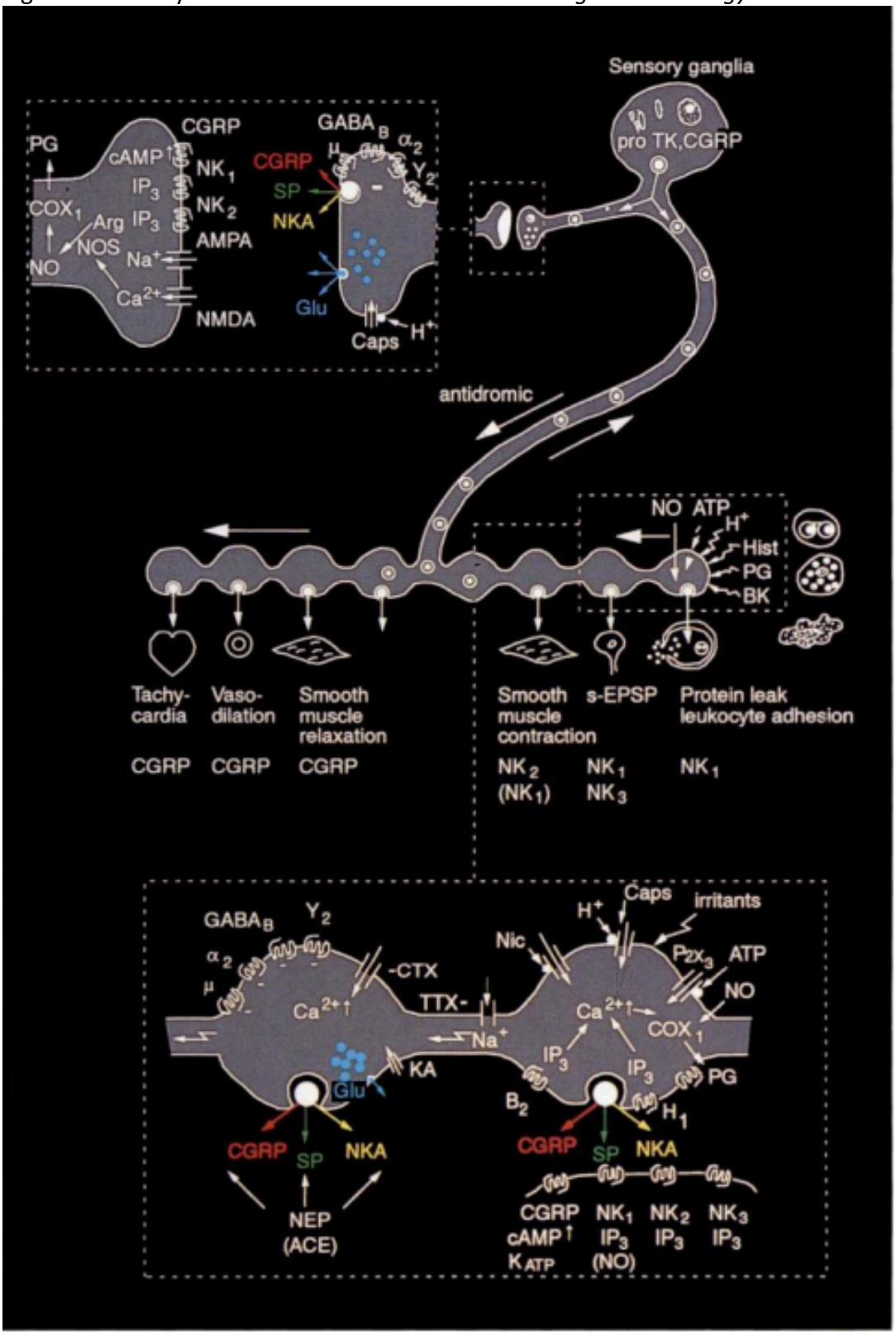


NPY caused vasoconstriction also in man (healthy human volunteers) leading to blood pressure elevation (Dr G Ahlborg collaboration). Release of NPY could be measured upon sympathetic nerve stimulation of the pig spleen as model organ, preferentially at high stimulation frequencies. In humans systemic NPY levels rose, indicating release, especially upon heavy physical exercise as a means of activating sympathetic nerves. Furthermore, NPY plasma levels were elevated in patients upon more severe heart failure, a known condition with strong sympathetic nerve activation (Dr J Hulting collaboration). Pharmacological depletion of NA levels by reserpine also reduced tissue NPY levels in certain organs. Furthermore, alpha 2 receptor blockade increased nerve stimulation induced NPY release suggesting that NA was controlling (inhibiting) NPY release. After NA depletion by reserpine, NPY release was enhanced and exceeded resupply with time leading to tissue depletion. As mentioned, reserpine treatment led to long-lasting depletion of NPY in certain tissues including vascular nerves and heart but not in vas deferens. This tissue specific depletion by reserpine of NPY could be prevented by reducing sympathetic nerve activity. After such procedures, in the absence of NA with enhanced NPY release also a long-lasting, presumably NPY mediated, sympathetic nerve stimulation induced vasoconstriction could be demonstrated in several vascular beds (spleen, kidney, skeletal muscle and nasal mucosa) and various species (pig, dog and cat). After NPY receptors were cloned by other groups ,it was determined by NPY peptide fragments that the vasoconstriction was via Y1 receptor in most blood vessels except the pig spleen where Y2 receptor seemed to dominate as for the prejunctional effect in vas deferens. One specific blood vessel which had a slow long-lasting contraction even in control conditions in vitro upon electrical stimulation was guinea pig vena cava where NPY could be involved as transmitter. NPY in addition inhibited the vagal tone of the heart, mimicking the effect of sympathetic nerve stimulation (Potter) presumably via Y2 receptors (Björkman) which may be of relevance for stress induced arrythmias.

IV) Multiple peptides in capsaicin sensitive sensory nerves

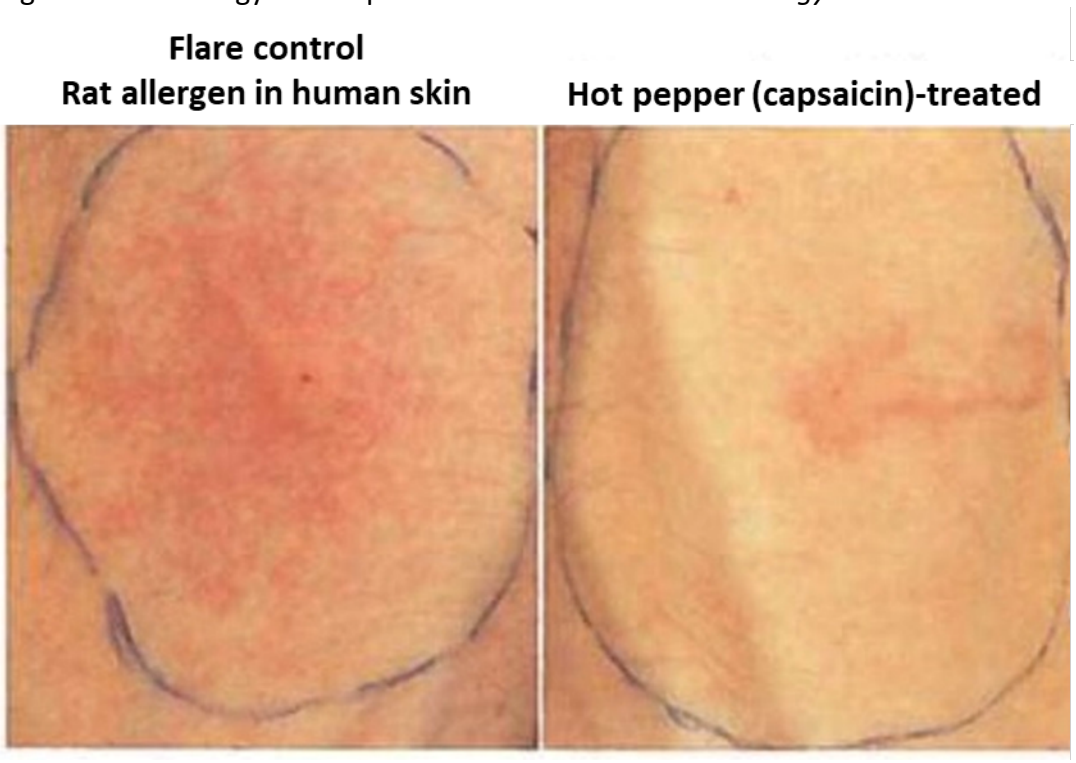
After the discovery of SP in sensory nerves, a tachykinin analogue neurokinin A (NKA) was isolated by other groups) from the same peptide precursor. The biological activity of tachykinins were found to be mediated by neurokinin (NK) 1,2 and 3 receptors. The dominant receptor for plasma protein extravasation was NK1, while contraction of visceral smooth muscle including bronchi and urinary tract was NK2 (Fig 3).

Figure 3. Sensory neuron transmission. From *Lundberg Pharmacology Review 1996.*



Later another substance, Calcitonin gene related peptide (CGRP) (isolated by Amara & Rosenfeld) was discovered in sensory neurons and found to coexist with tachykinins to a large extent in various sensory ganglia (Fig 3). CGRP was a potent vasodilator, stimulated heart muscle and relaxed certain visceral smooth muscle. By using specific pharmacological antagonists, a picture has emerged that sensory nerves can release multiple bioactive peptides from peripheral branches causing plasma protein extravasation, smooth muscle contraction/or relaxation and vasodilatation. Central Transmission of irritation can probably be enhanced by CGRP. These sensory nerves are polymodal and can respond to a variety of chemical irritants, low pH, heat, allergen and inflammatory mediators. A classical activator is capsaicin, the pungent agent in hot peppers. Capsaicin and a super potent analogue, resiniferatoxin, binds specifically to a subpopulation of sensory nerves activating an ion channel (Julius et al.) and causes burning sensation and release of multiple bioactive peptides. Higher concentrations of capsaicin in addition desensitizes nerves and cause degeneration and long-term peptide depletion. Further after capsaicin treatment in high concentrations the cigarette smoke irritation (bronchial protein extravasation in rats) and allergen evoked flare (vasodilatation) in the human skin (Fig 4) are prevented suggesting SP and CGRP involvement, respectively. Further capsaicin and CGRP are potent vasodilators of human cerebral vessels.

Figure 4. Skin allergy with capsaicin. From *Lundblad et al Allergy 1987.*



Since recent data from several pharma companies show that oral CGRP receptor antagonists or systemic CGRP antibodies (including Emgality from my Lilly career, Fig 5) reduce number and severity of migraine attacks in humans; sensory nerve mechanisms involving CGRP release may be of key clinical importance.

Figure 5 CGRP antibody structure, Emgality, Lilly

A summary of scientific findings in the cotransmission area (I-III) has been published: Lundberg, J M Pharmacology of cotransmission in the autonomic nervous system, Pharmacol reviews, 1996, 48,113-178.

V) Nitric oxide as mediator

Nitric oxide (NO) is a potent vasodilator (being the endothelium derived relaxant factor, EDRF, Furchott). When NO is inhaled it can reverse pulmonary vasoconstriction upon eg. endotoxin shock. NO is also a component of cigarette smoke causing increased blood flow in airway mucosa. NO can be formed in the body by various nitric oxide synthases (NOS) and is exhaled locally

produced from airways especially nasal sinuses. NO concentration in exhaled air is increased upon human allergic asthma and when saliva (nitrite) is reaching the acid pH of the stomach.
The observation of increased NO levels in exhaled air of asthma patients led to patent applications and formation of Aerocrine, a biotech company together with the Dr Lars Gustavsson group from KI Physiology department. This started ability to diagnose allergic asthma point of care.

Neuronal NOS seems to be present in certain autonomic nerves (potentially with VIP, acting as a "gas transmitter") and may contribute to autonomic nerve vasodilatory responses or smooth muscle relaxation including penile erection. (Figure1) The relative contributions by VIP and NO to autonomic responses need to be further studied using more powerful specific pharmacological agents.

VI) Endothelin

Endothelin is a potent vasoconstrictor peptide (Dr Yanagisawa) in experimental animals and humans, derived from endothelial cells. Increased plasma concentrations of endothelin can be seen upon experimental endotoxin shock. A pharmacological endothelin antagonist protected against pulmonary vasoconstriction by endothelin and endotoxin. This agent is now used to treat pulmonary hypertension.

VII) Thoughts and learnings from a dual academic, biotech and Bigpharma career

Academic scientists have a complex task combining research, teaching and generating data applications with relevance for society. During my career these tasks have increasingly become more related and to obtain intellectual property and start biotech companies based on government funding have become encouraged (in contrast to the past). Based on the expertise and research content, the Pharmacology department has always been closely associated with the pharma industry, either for advisory roles, collaboration partner or future career opportunities.
Academic positions have changed from doctorand scholarships to fewer more secure research positions including during thesis work. Professorships are not always openly applied for (as in past) with national or international competition, but it is largely up to the university to promote professors. This has vastly increased the number of Professorship roles, some of which in the 1980s were dedicated for female applicants (Tham professorships).

Personally I wanted to stay at the KI, having an excellent research group and collaboration network also being recognized of coming from a world renowned medical university even if I applied for and got professorships in Pharmacology at other Swedish universities.
In my discussions about my future with leading KI representatives I got the comment that "hungry wolves hunt better". There is always a question whether it is beneficial to become professor with a tenure position and department resources and obligations at a very early stage of the research

career? When I got the early medical research council position it benefitted my time and resources to do relevant research, which was further substantiated when I finally became professor of neurotransmission research (Fig 6) (Appendix 1).

Figure 6 Professor celebration

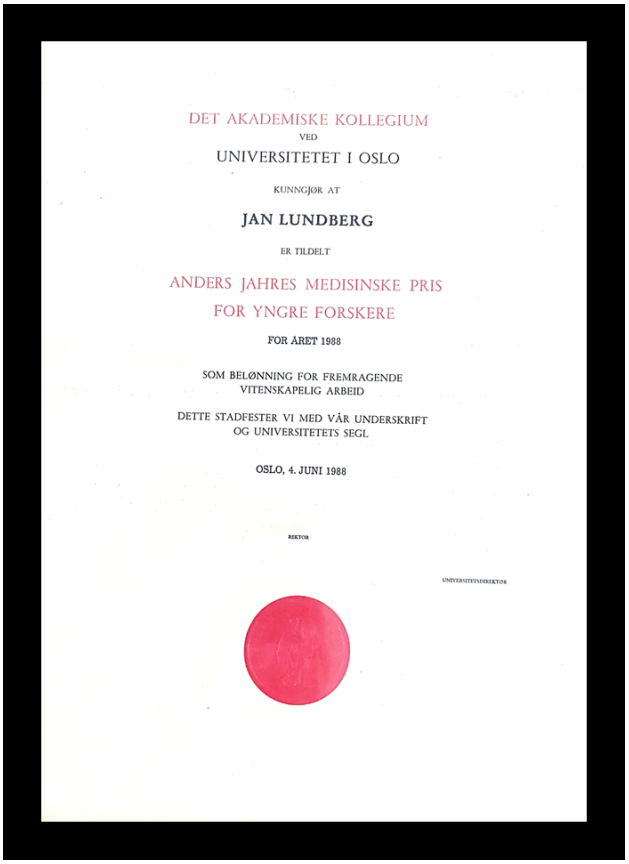
Scientists with broadly related research topics or methodologies are gathered at KI departments. In theory these scientists should interact in a collegial way, potentially collaborate and share teaching obligations and common resources including PhD students. Human nature does not always allow optimal interactions, however, considering that scientists often are not so collaborative being very competitive individuals. In addition, career advances or grant application success are sometimes hindered if too many senior scientists are coauthors.

Personally, I have prioritised collaborations to access peptides or antibodies as well as doing human experiments and accessing patient material. Regarding PhD students or guest scientists, it is obvious that my group benefitted hugely from talented individuals that gave their energy, intellect and time doing experiments and writing papers (Appendix 2). It has been a great joy to see sometimes relatively unexperienced individuals take on new tasks and grow into mature

scientists with their own careers (several becoming leaders in academia, in the clinic or in the industry).

Science evolution needs a constant influx of new ideas and efforts. I had the benefit to be exposed to and interact with international scientists at conferences at an early stage. In fact, I was invited to be chairman of an international peptide symposium before my thesis defense. These congress presentations can be sometimes painful if your data are novel (controversial) and, there is in addition an English language barrier (particularly when need to deal with sometimes aggressive culture in Question-and-Answer sessions, but it is a natural part of a scientist's maturation to also question your own data and to do the right controls (including repeat experiments) before publications. Scientific awards and recognition are highly motivational, and I have been fortunate in that area with the Jahre (Fig 7) and the Fernström prize.

Figure 7 Jahre prize


DET AKADEMISKE KOLLEGIUM
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UNIVERSITETET I OSLO
KUNNGJØR AT
JAN LUNDBERG
ER TILDELT
ANDERS JAHRES MEDISINSKE PRIS
FOR YNGRE FORSKERE
FOR ÅRET 1988
SOM BELØNNING FOR FREMRAGENDE
VITENSKAPELIG ARBEID
DETTE STADFESTER VI MED VÅR UNDERSKRIFT
OG UNIVERSITETETS SEGL
OSLO, 4. JUNI 1988
REKTOR
UNIVERSITETSDIREKTØR

One sometimes forgotten aspect of doing intense research is the need to relax and do completely other activities. I choose to do physical exercise (marathon cross-country skiing; the Vasa race 28 times completion over the years, Fig 8). I also have a very supportive family with my wife (Ingeborg, Fig 8), four children (Erica, Rebecca, Martina and Nils) and seven grandchildren.

Figure 8 Vasarace

Many aspects of my academic learnings I could also translate into my industry career (Appendix 1). Collaboration and teamwork is paramount in the complex and highly regulated and controlled industry activities to discover and develop a new medicine. My training as pharma science consultant and expert in the Medical Product Agency for approval of new medicines helped me to prepare for my next career step.

In 1995 I was contacted by several head-hunting search firms for senior research roles in the pharma industry. I was on my way for an interview with Roche in Basel, Switzerland when Astra also contacted me for an interesting role. I had worked as a consultant to both the Astra Draco and Astra Hässle divisions for some time so for me that was a more familiar environment with people like Dr Bengt Åblad at Hässle cardiovascular area. I was called to the Astra head office at Södertälje for an interview and to meet Håkan Mogren, the Astra CEO. After the interview, I was offered a new role as Astra preclinical research head with mandate to initiate and implement new drug-hunting technologies (forming so called competence centers for High throughput screening, Structural chemistry, Biological agent discovery and development, transgenic animals and informatics) and to coordinate related research applications across the various product companies.

An exciting task in 1996 was to fly to Boston, buy land and start a new research site with the goal to eradicate Helicobacter pylori, a key pathogen for duodenal ulcers. An area where the Astra drug Prilosec, a proton pump inhibitor, had great success in addition to treating gastro-esophageal reflux disease becoming the largest selling pharmaceutical in the world! This income

for Astra provided great resources, at a level unheard of in academia. Regarding my KI professor role, the new KI president Hans Wigzell, said my career change to Astra could be good also for KI (with potentially new collaborations) and he gave me a two-year test period, keeping my position (leave of absence). After my transition, current PhD students and recent PhDs got free access to my lab and resources to complete their activities on their own studies and papers (Appendix 2).

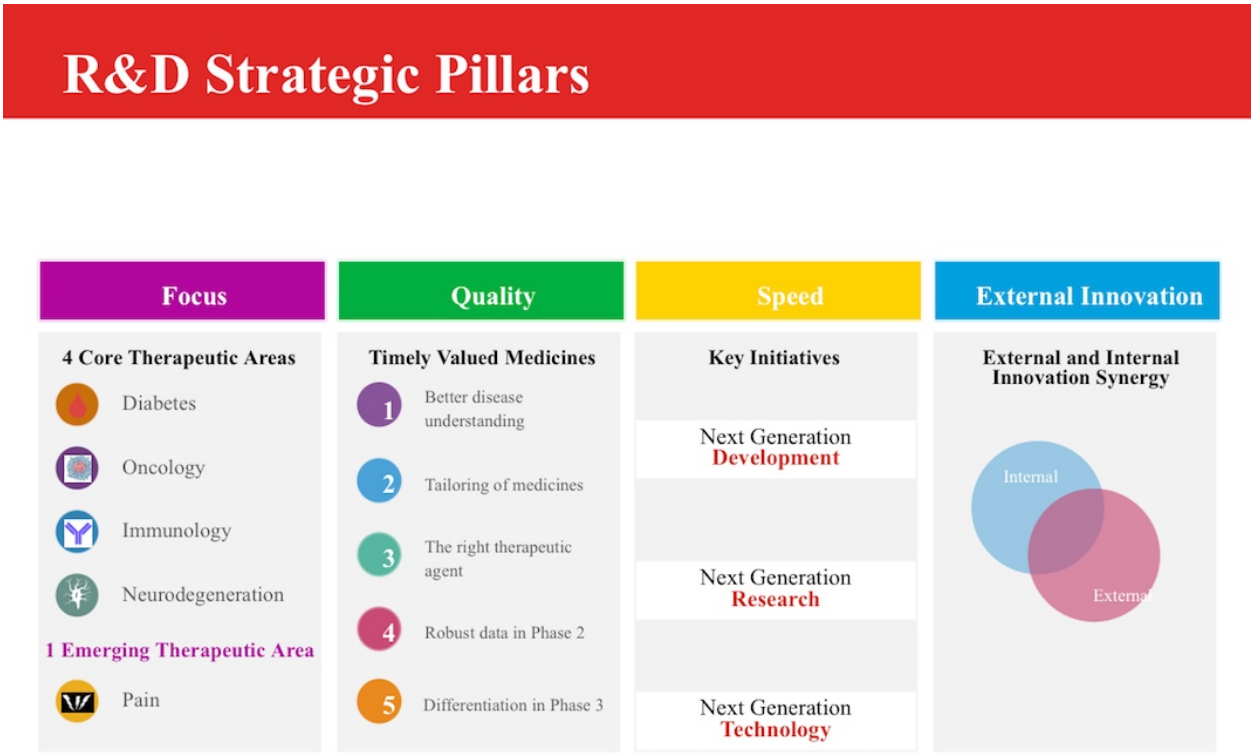
Then in 1999 came the AstraZeneca merger, a time of severe unrest! I was surprisingly appointed by the new AstraZeneca CEO Tom McKillop to global head of discovery research and to lead the research integration between the large organisations of Astra and Zeneca (despite having considerably less industry experience than many senior Zeneca colleagues). This was a major challenge involving support/steer from consultants (McKinsey) which had their agenda and done mergers before. They talked about the need to make merger synergies. Synergy reminded me scientifically of the effects from VIP and Ach on salivary secretion: 1+1=3. But they said I was in the wrong area of mindset: 1+1=1.5 in the merger world, one goal is to financially reduce costs and infrastructure of the combined organization to save money in addition to create more products! Consequently, several sites were closed down and some key people offered to move. My learnings after some years of recovery were still that the road ahead for large Pharma organizations was long-term science & Innovation (Lundberg & Reilly, Drug Discovery Today, 2009).

The turbulence settled with time but after 10 years in this executive role with thousands of scientists at many sites globally, I was approached in 2009 by an executive headhunter Sandro Franchi from Eli Lilly, a large US based pharma. This was an exciting new opportunity as President of Lilly Research laboratories (Head of both research and development) based in Indianapolis, Indiana in the US Midwest south of Chicago. The employment conditions were very attractive, and I felt like a scientist (and ex-hockey player) that had just got an offer of an "NHL level employment contract."

Lilly was well known for large R&D resources with a leading neuroscience portfolio including drugs like Prozac and Cymbalta as well as new approaches for Alzheimer's disease. The effort in diabetes where Lilly had been a pioneer with insulins had been scaled down, but new areas were emerging like cancer and inflammation. Dr John Lechleiter, the CEO and chairman of Lilly's board was my new boss, and he uniquely had a scientific background with deep R&D understanding and experience, unlike most other pharma CEOs which have a sales & marketing finance background.

After some initial late-stage hiccups in psychiatry (failures are unfortunately common in this area with less than 10 % overall success rate), Lilly under Dr Lechleiter still continued to support pipeline programs in other areas, and soon a unique series of two major new products could be launched every year, often in combination with new diagnostics, up to my retirement in 2018 and beyond from internal pipeline and external collaboration opportunities. This track record of productivity is highly unusual in the pharma industry. A strategy across Lilly named "Timely valued medicines to patients" was used with great success (Fig 9).

Figure 9 R&D strategy in pharma



This strategy included raising the bar for differentiation to satisfy demands of payers, providers and patients before progress of programs along the R&D value chain. With time this created blockbuster (multibillion dollar) products like Trulicity and Jardiance for type II diabetes, Taltz for psoriasis and ankylosing spondylitis and Verzenio for metastatic breast cancer. This product surge enhanced Lilly's Share price several fold and I was also pleased to support the R&D and successful registration of Emgality, a CGRP antibody (Fig 5) for migraine prevention, closing the loop from academic career KI research to human patient benefit.

Figure 10.

In addition, the future Lilly pipeline, created to a large extent by new R&D investments like in San Diego, (Figure 10), has promising novel projects for Inflammation (Mirikizumab), Diabetes and obesity (Tirzepatide) and Alzheimers (donanenumab).

Overall, transition from academia to industry has given me extraordinary motivational opportunities to really do large scale translational research and to influence discovery and development of 25 new medicines in areas of unmet medical patient need using my scientific and medical training.

After my retirement from Lilly in 2018, I have used my long experience with medical research and continued my research interest as board member and advisor (Appendix 3).

Appendix 1 My education and career advances

1973-77 University of Gothenburg Medical school

1978-1981 PhD student Karolinska Institute

1981 PhD thesis KI (Pharmacology and Histology departments)

1988 Anders Jahre Scandinavian Research award for young scientists

1984 Medical research council research position

1987-90 Läkemedelsnämnden, Läkemedelsverket, new drug approvals in Sweden

1988-95 Medical research council prioritization committee for physiology and pharmacology

1990- Chairman of PhD and Docenturnämnden, KI

1992 Professor in neurotransmission research,

1993 Fernström's research prize

1992 Highly cited author, Current content "of Nobel class," A citation perspective on high impact research authors" Garfield,A and Williams D.

1995-1999 Preclinical research head Astra AB

1999-2009 Global head discovery research, member of executive committee, AstraZeneca.

2000-2005 Strategic research foundation member

2000 European union special pharma /academia program, Innovative medicines initiative (IMI,)core founder group

2005 Honorary doctor, Pharmaceutical faculty, Uppsala universitet

2009 -2018 President Lilly research Labs (Global head research and development) member of executive committee (Contributed to 25 new medicines in aggregate during industry career, 2005-2018)

20015-18 National institute of health USA. Accelerating Medicines Partnership (AMP), committee for advancing academia and industry collaboration

2017 Prix Galien award nominee for best pharmaceutical product (on behalf of Lilly)

1995-2018 Hever group member, fostering collaborations between big pharma, academia and regulatory authorities globally.

Appendix 2 PhD students and guest scientists at my lab (Appendix 2)

Contributing to more than 500 publications in referred journals. They were supported by excellent lab technicians: Anette Hemsen Mörtberg, Margareta Stensdotter and Carina Nihlen at Pharmacology Dept and Waldtraut Hiort and Gun Norell at Histology dept

PhD students and guest scientists

Lars Lundblad (ENT,KS)

Claes Roland Martling (Anestesi & intensivvård,KS)

Anders Rudehill (Neuroanestesi, KS)

John Pernow (Farm, KI) (Prof Cardiology)

Anders Franco-Cereceda (Farm, KI) (Prof Thoracic surgery)

Eddie Weitzberg (anestesi KS) (Prof Anestesiology)

Kjell Alving (Farm,KI) (Prof Pharmacology)

Cecilia Fornhem (farm,KI)

Agnes Modin (farm,KI)

Per Stjärne (ENT,KS) (Prof ENT)

Johan Rinder (ENT,KS)

Anette Hemsen (Farm,KI)

Olof Larsson (Farm,KI)

Richard Malmström (Farm,KI)

Jon Lundberg (Farm, KI)(Prof Pharmacology)

Xiao-Xing Hua (Farm,KI)

Ya-Ping Lou (Farm,KI)

Silvain Lacroix (Farm KI,Geneva)

Regis Matran (Farm KI, Paris)

Alois Saria (Farm KI and Graz)

Arpad Szallasi (Farm, KI and Budapest)

Rainer Gamse (Basel)

Philippe Delay-Goulet (Paris)

Kyowa Hakko (Japan)

Björn Edwall (Farm,KI)

Serge Auberson (Geneva)

Elisabeth Änggård (Danderyd's hospital)

Appendix 3 My Board and advisory positions 2018-current

Ardelyx inc (San Fransisco)

Metabolon (Raleigh, North Carolina)

Imaging analysis group (London)

Betagenon (Umeå)

Anocca (Södertälje)

Ferring (Geneva)

Tuberculoisis alliance (Charity, New York) ended 2021

My Advisory roles

Rehnman investment (Stockholm)
Sigrid Therapeutics (Stockholm)