Experiences of therapy with haptens

by Peter Cornelius
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In the middle of the 1970’s I had a visit from a businessman who wanted to show me some isolated hapten preparations which he intended to sell in Germany.

And so it was that I first learnt that haptens which have been isolated from pathogens and are polysaccharide in character can also be used therapeutically. However, he could not provide any clues as to the indications for which their use was to be recommended. He could only report that the remedy Haptenovacuna was prepared from a strain of bacillus which was cultivated in the damp environment of the nasal passages and got its name (vacuna = cave) because it was useful for the nasal sinuses. Furthermore, that his own psoriasis had been healed with Polipse (= polysaccharido de pseudomonas). Polipse is also attributed with an effect similar to cortisone without having the damaging side-effects typical of steroids. He had tried to have this effect of Polipse (which he had discovered to be positive on himself) tested by the University Dermatology Clinic in Hamburg in clinical trials. It never reached the trials stage as the costs of the fee demanded and samples required would have far exceeded his financial capabilities. And so then with the aid of proving I began to determine in which cases the therapeutic use of these preparations would seem sensible. I want to report here on my current state of knowledge, made up mainly of my experiences and the study of Jan Klein’s Lehrbuch der Immunologie [Textbook of immunology]. I could not find any helpful insights into haptens in other books on immunology.

What are haptens?

The concept „hapten” was introduced to immunology by Landsteiner in 1923. It is derived from the Greek word „haptēn” which means to stick, as haptens stick to the carriers of antigenic features. These carriers which are linked to haptens are known as conjugated antigens. The molecular weight of a hapten should (according to Jan Klein) be less than 4000 Daltons; other authors suggest that it could be even less than 1000 Daltons, whilst J. Hartmann determines a molecular weight of approx. 5000 Daltons for the hapten deriving from Brucella melitensis (polysaccharide B).

Haptens have two bonding valencies. In conjugated antigens one of these valencies at a time is bonded with the carrier. According to Jan Klein, if conjugated antigens enter the body, the other valencies of their haptens can only be taken up by the B-cell receptors (=& BCR), the B-lymphocytes which originate in the equivalent of the bursa.

In contrast to the haptens, which in their isolated state do not have any antigenic features, the carrier molecules must have a minimum size of 4000 Daltons in order to develop antigenic features. Highly active antigens can reach sizes in excess of 100,000 Daltons. These carriers of antigenic features can only be bonded by the T-cell receptors (=& TCR), the T-lymphocytes which are formed by the thymus. According to Jan Klein, only when a conjugated antigen is bonded simultaneously by its carrier part to a TCR and by its hapten part to a BCR can the B-cell form and deliver the specific antibodies. If there are no haptens present, the free carriers are in fact also bonded by the TRCs, which causes them to react by inducing inflammation. However, bonding to the B-cells in this way is not possible and so the formation and release of antibodies cannot take place.

If haptens are not present, the second half of the immune response is therefore inevitably blocked and inflammations of this type cannot be stopped: they become chronic and - as it is hardly possible to discern an acute phase - are labelled as primarily chronic. Very different types of substances can act as haptens, e.g. lipids, lipoprotides, nucleic acids or polysaccharides. Haptens which are found in germs which induce illnesses are nearly always polysaccharides which are specific to the particular pathogens or groups of pathogens.

Meanwhile it was becoming clear to me that Polipse as a hapten from the pathogen Pseudomonas aeruginosa (previously known as Pyocyanus) bonds with the carriers from this pathogen and with other free antigens which have certain similarities. This means that by substituting haptens which clearly must somehow have gone astray, the free carriers are again changed back into conjugated antigens. As they do so, they can now bond again with BCRs and the release of antibodies which was not possible before is enabled; or if there are not already some lying ready in memory cells, they begin to form.

The prompt effect, frequently experienced in a wide range of cases where haptens are used therapeutically, testifies to the fact that isolated carriers, each bonded to TCRs and thus to T-lymphocytes, must have been present and were responsible for the inflammation. T-carrier complexes of that kind were also being overcome in this manner, whereupon the inflammation, if it had been caused by these, could be stopped immediately by removing their primary cause.

These facts led me to label haptens being used therapeutically as antigen absorbers. Thus new therapeutic possibilities were opened up in the treatment with haptens of illnesses which depend on the presence of isolated carriers. In the case of the dealer, his psoriasis must have been being maintained by carriers from Pseudomonas or by other isolated antigens which could also bond with the Pseudomonas haptens and therefore could be cured with this hapten.

As in practice I have not met any further case of this disposition, I expect that such a situation occurs only infrequently, which is why a clinical study based on this question alone would probably have led to a disappointing result. Supposing that you had treated 1000 randomly selected psoriasis patients with Polipse and achieved a cure in three of them, you would surely not attach any statistical relevance to this 0.3 per cent and would therefore forget the trial. In actual fact
the test would have shown that only in these three patients was there an indication for the use of Polipse, and that where the indication was present there was also 100% efficacy.

This raises the question of how isolated carriers can cause loads of this type. If we understand that polysaccharides, e.g. starch, are split by the enzymes in the digestive tract and that if required the glycogen in the muscles can be split into monosaccharides, it can be assumed that polysaccharide-like haptenic carriers from microbes can be destroyed by enzymes. Thus it is to be expected that microbial carriers can be released from infected material which reaches the digestive tract or develops there, even when the pathogens have been destroyed by heating the infected or contaminated food or by treatment with antibiotics.

In order to avoid destruction of haptenic compounds being used therapeutically in the digestive tract, as far as possible they must therefore be administered parenterally, i.e. through intramuscular or subcutaneous injections. Hapten preparations in the form of drops probably have a better effect if they are rubbed in percutaneously (e.g. into the inside of the elbow) than when given orally, which exposes them to the digestive enzymes.

Through proving it was possible to optimise accuracy in setting indications for the use of haptenic procedures such as electroacupuncture according to Voll (= EAV), kinesiology or even radiesthesia require individual talents which up to now it has not been possible to record scientifically and which certainly can only be partially learnt. An understanding of these methods cannot be expected of doctors who have only been trained in medical schools. However, these testing procedures cannot and do not have to be explained here, as extensive literature about them exists and it can be assumed that they are well-known in circles concerned with natural healing.

Thus from the purely scientific point of view, in clinical examinations the indication for the use of haptenic compounds can only be established through therapy trials. Only from an observed treatment result with a hapten as antigen absorber can the conclusion be reached later that the particular problems must have been caused by isolated carriers which were again bonded by the haptenic compounds which had been administered. One hapten can therefore function like a differential therapy tool which has no possible effect when indications are absent, i.e. when the problems are not caused by free carriers which can join to the given type of hapten. It therefore goes without saying that randomised double blind studies are of as little use in determining the effectiveness of haptenic compounds as if you wanted to test the germination of seeds by scattering them on a paved area. If in this case only three seeds in a thousand sprout, it does not mean that the germination rate is only three per thousand: instead it shows whereabouts enough humus had collected for the seeds to be able to germinate.

Thus there remains for the time being only the experience of obtaining findings about opportunities for the therapeutic use of haptenic compounds.

After a period of observation of approx. 25 years, during which I have used over 3000 ampoules of haptenic compounds, three main areas of indication have become apparent to me. These have to be looked at more closely in the light of clear-cut examples, in order to find clues as to when hapten therapy can be tried out with a good chance of success.

A. Complementary, post-antibiotic treatment with haptenic compounds when problems persist although the infections have already been overcome

Case 1
In July 1988 the father and son of family K. were taken ill with persistent diarrhoea. Examination of the faeces for pathogens showed that in both patients it was likely that massive intestinal candidiasis was the cause.

After antimycotic treatment with nystatin, the son soon recovered fully. The father, Mr E.K. (then 49 years of age) not only appeared not to be responding to this treatment, but he also started to have pain in the joints of the spinal column. A further course of treatment with amphotericin did not bring about any improvement. However examination of the faeces showed that the candida colony had for the most part disappeared. At this point he came to my practice. He received an injection of 0.1 mg candida haptenic compounds. This at once ended his problems, both of the intestine and of the spinal column.

Case 2
The tonsil abscess of a patient was treated by operation and administration of antibiotics. The tonsils had healed well, but the patient felt more shattered than before. Doubtless she had swallowed pus when the abscess had been incised. A few minutes after the injection of 0.1 mg streptococcus haptenic compounds her condition improved visibly, and a second injection of the same on the following day restored her completely.

Case 3
In September 1999 40-year-old Mrs C.S. came to see me. Six months earlier she had been operated on for an umbilical hernia. As a result of this operation she had contracted streptococcal septicaemia. It had been possible to control this with an intensive course of antibiotics, but the patient had been suffering since that time from constant pain in the intestines with frequent diarrhoea as defined by post-antibiotic colitis.

After an injection of 0.1 mg streptococcus haptenic compounds, on the following day she had violent stomach pain which lasted for an hour; afterwards her problems were much reduced. Three days later she had a short episode of stomach pain with sickness. This finally brought Mrs C.S.’s problems to an end. This success shows that in this patient isolated carriers from streptococci must have been responsible for the persistent pain.
As everybody knows, similar conditions can also be caused by overgrowth of the intestinal flora with the multi-resistant germs of Clostridium difficile. In such cases a course of treatment with the nosode Clostridium difficile, which sometimes has to be supplemented by treatment with SANUKEHL Serra. The company Sanum-Kehlbeck GmbH & Co. KG, Hasseler Steinweg 9, D-27318 Hoya/Germany, has proved successful. The therapy is evidently able to stimulate the immune system so specifically that these pathogens are cut down to size.

Similarly, patients with problems which persisted after antibiotic treatment of staphylococcal infections had to be given staphylococcus hapten, coli hapten after antibiotic treatment of coli infections, and proteus hapten after proteus infections.

B. The treatment of non-infectious pathogenic alimentary toxicoses

As there is no reason to accept that botulism is the only non-infectious pathogenic alimentary toxicosis, it has to be reckoned that problems can also be caused by taking in other pathogens orally, even if these had been destroyed by boiling or pasteurisation.

Case 4

On 4.7.1995 Mrs N.L. presented herself for the first time with an acute attack of rheumatism. I had already known her for a long time: at that time she was 33 years old. That morning she had woken up with completely stiff, painful finger and toe joints. Her whole hands and feet were red and thickly swollen with oedema.

On the previous day she had eaten poultry, which I presumed to have been contaminated with tuberculin. Just five minutes after she had received an injection of 0.1 mg BCG hapten, the swelling began to reduce significantly and her fingers were free of pain again. I assumed that the cause of this attack of rheumatism was contamination of the alimentary canal by tuberculin toxins, from which the carriers had been released in the digestive tract.

For as long as I was still in contact with this patient, she never had this type of problem again. The assumption which had prompted me to try the treatment was afterwards confirmed by the good results of the treatment.

Case 5

On 6.3.1998 Mrs E.H. (then aged 73) asked me to make a home visit. She had suddenly been taken ill with nausea, sickness, diarrhoea and such powerful dizziness that she could not stand up. Previously she had probably eaten some sheep’s cheese imported from the Balkans. The injection of one ampoule of 0.1 mg brucella hapten brought this state of affairs to an end in five minutes, which leads me to conclude that this cheese was contaminated with Bang (brucella) antigens.

The observations described under A and B can perhaps only be explained by means of the hypotheses proposed above. It is possible that despite treatment with antimycotics or antibiotics (as in cases 1 to 3) antigens remained in the body, or that heat-resistant antigens entered the body (as in cases 4 and 5) from pathogens which exist in foods, even if the pathogens had been destroyed by pasteurisation or boiling. The carriers must have been released from such antigens, which were originally still conjugated, when they were destroyed by the action of enzymes on the hapten in the digestive tract. As these carriers had only just been bonded by the TCRs in the lymphoid tissue of the wall of the intestine without the B-cells having been able to render them harmless, in the patients concerned the lost hapten had to be substituted as quickly as possible in order to enable the otherwise blocked second half of the natural immune defence by the B-lymphocytes to take place.

In cases 4 and 5 there were clearly enough specific memory cells in which the antibodies required were already being held on standby. Therefore antibodies could immediately bond the TCR carrier complexes with the matching hapten doses and eliminate them. This also immediately stopped the inflammations which were caused by T-cells, because the primary cause was removed.

If antibodies and memory cells first have to be formed, the process will naturally take longer. In individual cases the body can react by raising its temperature in order to speed up this second half of the immune response.

If then the hapten dose results in a raised temperature, it is not a question of it being an undesirable side-effect; rather, it is a part of the desired and necessary main effect. On no account should such a fever be combated with an antipyretic or hindered in any way, but rather it should be supported by supplying more heat.

C. Hapten as a supplement to nosode therapy

Non-infectious pathogenic toxicoses, or those which are no longer infectious after treatment with an antibiotic, which have been caused by free carriers and which cannot be eliminated immediately by a corresponding dose of hapten or overcome by enzymes, can turn into chronic conditions quickly or even slowly and gradually according to the disposition of the toxicosis concerned. This probably happens by the coupling of the TCR carrier complexes with endogenic elements of tissue which contain components which can serve the carriers involved as a hapten substitute. In this way the contact points with which they should link to their haptenes are already used up. Therefore these chronic conditions are no longer accessible to primary hapten therapy.

1 By ‘residual toxicoses’ we mean contamination which can result from a great variety of infections and which can in particular remain when spontaneous healing during the acute phase of the illness is prevented by remedies which inhibit inflammation and reduce fevers.
Together with pollutants coming from the environment, the TCR carrier complexes fixed in the mesenchyme and other residual toxicoses in this way lead to what Dr Reinhard Voll called slackening of the mesenchyme. Many contaminations of this sort can be overcome by means of nosodes of the same type, and the homeopathised pollutants concerned can be overcome with suitable complementary therapy.

By treating with nosodes and homeopathised pollutants there may be initial aggravation. This happens because these poisons and pollutants which have been mobilised by the isopathy must be channelled through the circulation before they can be excreted.

If such initial aggravations do not ease in two to three days, this mostly stems from the fact that there are TCR carrier complexes in the mobilised toxins which are freed as a result of the specific fields of tension of the nosodes from the connection to endogenic tissue and thus are re-activated. In this way the illness which had drifted into the chronic state is brought back to the acute state. In these circumstances, exactly as in the beginning, the second half of the immune response - which is still blocked by the lack of suitable hapten - can only be set in motion again by the substitution of these very hapten. This is the most natural and the quickest - perhaps even the only - way to true healing.

However, it is unfortunately not possible to count on the fact that every patient will take note of the circumstances of the aggravation caused by the hapten and will report on it. Therefore during a course of nosode treatment I carry out a control test before every injection; for when a hapten is needed, the combination of nosodes and complementary remedies which would be used in turn will not fit until this hapten has been given. As a rule, the treatment can then be continued the next day according to plan.

With true healing there is also the formation of memory cells, which get their name from the fact that they hold the method of production of the antibody specific to a particular antigen in their memory for use in the future.

Thus at the same time the creation takes place of a form of protection against new infection by the same pathogen which should not be underrated. In our time, when there is concern about the growing level of resistance in all the different pathogens against almost all antibiotic medicines, this could be of great importance.

Case 6
A man of approximately 30 years of age, who came into my practice in about 1983, was suffering from intense pain in the lumbar region of the vertebral column.

Following a CT scan he had been advised to have an operation on the intervertebral disc between the 4th and 5th lumber vertebrae. Before he decided on the operation, he still wanted to try alternative therapy. The testing showed contamination with tuberculium, as well as Teucrium scorodonia.

Immediately after the first injection the pains in his spine became considerably worse. This was remedied with a dose of 0.1 mg BCG hapten a few hours later. In this case a similar injection of BCG hapten was necessary on each day following the nosode injection. After the tenth and last nosode dose the patient was completely free of problems and did not reconsider having an operation.

Without the hapten the course of nosode treatment in this patient could not have been completed. An operation could probably not have freed the patient from his pain, as it is impossible to remove bacterial toxins by rough mechanical means. But this can certainly be done isopathically, that is in practice using the most delicate mechanical means - the substance-specific high tension fields of the toxins themselves, increased in stages by homeopathic dilution.

However all these concepts are familiar and absolutely obvious to us through their effects which we cannot ignore and which we can see all around us. Now the effects of homeopathic high dilutions have also been observed by many thousands of practitioners in many millions of cases throughout the world. All the same, some people (and they think that they are entitled to speak in the name of Science) consider that they are entitled to challenge these effects simply because they themselves have not yet made similar observations. But in any case they wouldn’t be able to make any observations, as they hardly know the name of even one homeopathic remedy, let alone its remedy picture, and thus would not be in the position of ever being able to select the right remedy.

Let us now try, with the help of physics, to make clear the working mechanism of the diluted substance: We know that, in addition to the energies which are familiar to us such as gravity and magnetism, there must exist energies of hardly conceivable power, especially in the realm of atoms. If you tried to rotate a large
object in the same way as an electron rotates around the nucleus of an atom, it would inevitably burst long before even an appreciable fraction of it had reached a speed of that magnitude.

The splitting of the double helix of ribo-nucleic acid which is a necessary part of cell division is also an enormously dynamic process. The spiral chains of genes unwind and then after the doubling rewind themselves with unimaginable speed.

But there are also energies which hold together molecules of the same type, so that liquids form round drops; over a period of time even solid matter of the same type can pass through a whole mass of rock and form a crystal in one place.

Such energies are described as „surface tension“ or „coherence“. They can act in a similar way to a field of tension between two condenser plates: we know that when the distance between the plates is doubled, the tension and thus also the voltage is doubled.

It is known that the negatively charged parts of raindrops collect on the upper surface of the drop. Here, as they fall, they are torn off into tiny droplets and remain opposite the now positively charged main drops which fall faster. As the distance between the negatively charged cloud of small drops from the positively charged cloud of large drops increases, enormous electrical fields build up and discharge by means of lightning and thunder.

From the observed effects of homeopathic high potencies it can be concluded that in the process of potentisation similar high tension fields are built up which are specific to the relevant substances.

Thus with every stage of potentisation of the D potencies, the distance between the particles will increase and therefore their tensions will also increase 2.15 times; in the C potencies they increase 4.64 times at every stage, and in the L potencies 36.84 times. You can imagine the energies which are released as being like unbreakable rubber bands which become increasingly thinner as the distance between the particles becomes greater, yet their tractive force increases at every stage.

Think of a magnetic field which surrounds a piece of magnetised iron and is specifically attracted most strongly to iron but also slightly attracted to metals which are apparently close to iron such as cobalt, nickel and manganese. So, too, diluted remedies appear to have the strongest effect on things which are similar to them (i.e. isopathic). However, they still have an effect on diseases which are similar in their symptoms to the effect picture of the substances used. In this way the homeopathic effects become comprehensible.

Let us look again at Case 6, which is an example of conformity with the natural law, such as could be observed over and over again in well over one hundred patients during a course of treatment with nosodes. The hypothesis discussed above is unequivocally confirmed by this conformity with the natural law - namely, that aggravations which can occur after the administration of any isopathic remedy and did in fact occur in this case, are brought about by the high tension fields of the isopathic remedies which are specific to the substances in question.

These aggravations can be explained by the fact that toxins of the same kind - which in chronic illnesses requiring nosodes consist of microbial carriers - are torn by the remedial high tension fields from these camouflaging bonds to endogenic substrates and thus are re-mobilised. Through this mobilisation they re-enter the circulation, which then results in the type of initial aggravations described.

If aggravations of the type described in Case 6 do not ease in two days, this means that the TCR carrier complexes which have been set in motion again in this way cannot be eliminated simply with the help of the complementary remedy. But they can often bond astoundingly quickly with the help of the corresponding hapten, exactly as would have been possible in the beginning before they could fixate, and thus be grasped by the B-cells. This is the quickest way to defeat them.

However, as can now be understood, this defeat has not yet been possible in the phase preceding the mobilisation of the TCR carrier complexes.

It is possible for the conditions described in A and B, which turn out to be acute inflammation caused by the T cells, to change slowly into chronic inflammation as described in C.

Therefore it can happen that in transitory phases like this, in which only one part of the toxins is fixed and another part is still circulating, a hapten must first be administered (as in A and B) in order to eliminate the still free TCR carrier complexes. However, since some of these TCR carrier complexes have already settled on the endogenic substrates, it is also necessary in the end to give additional treatment with the corresponding nosodes, possibly requiring repeated intermediate doses of the corresponding haptens.
The observed effects shed new light upon the etiology of primarily chronic and auto-aggressive illnesses. If TCR complexes have arisen with free carriers without a suitable hapten being present, it is conceivable that replacement structures similar to haptens must be found so that a complete immune response becomes possible.

The experiences lead us to expect that a whole range of antigens have structures like this, e.g. in the substance of the articular cartilage.

As already mentioned above, nucleic acids - among other things - can take over the function of haptens. Since it is possible to find antibodies against nucleic acids in autoimmune conditions such as systemic Lupus erythematosides, it would appear that they are being also used by some carrier types as a replacement for their missing haptns. In these cases, if the B-cells want to eliminate the antigens concerned, they have to grasp the endogenic structures which are replacing haptns. If such autoimmune conditions have occurred and if healing is to be possible, then one has to look not only for the nosodes of the obliterating carriers but also nosodes of the type where the auto-antibodies which have formed are also preserved.

The B-cells have a primary immuno-tolerance towards endogenic substrates. Initially, therefore, antibodies to those tissue components which are bonded to TCR carrier complexes are suppressed. The cause of the inflammation which starts from these complexes remains unconnected to that. Therefore this type of inflammation is known as primarily chronic. But if suitable haptns are administered immediately at the beginning of the treatment, the positive reaction to treatment of this type of illness shows that this condition very probably also began with an acute condition and therefore only became chronic later.

If we are dealing with carrier TCR complexes which come from pathogen types which as a rule only cause reactive arthritide, these can apparently also be overcome or secreted spontaneously. Possibly because these find enough substances to be a replacement for haptns which are not so specifically endogenic that the antibodies needed for their defeat must be auto-aggressive, or because they can be released as enzymes, whether by endogenic enzymes or those supplied by remedies such as Bromelain, pancreatic enzymes or Serrapeptase. In general it is not possible to rely on the possibility of these so-called reactive arthritides being overcome spontaneously. That is to say, many patients with these arthritides have to be given the nosodes of the corresponding pathogen types. Because of the nosodes, inflammations of this type also begin to ease, whereas in other people they would only appear as reactive, since they disappear without any form of treatment. If, on the other hand, free carriers of pathogenic mycobacteria or streptococci are responsible for the starting of inflammations, their TCR carrier complexes appear to remain invulnerable to the immune system without the missing haptns being substituted over a long period of time, and for this reason they are not classed as only reactive.

Dr Nieper reported that in MS patients the medullary sheathes of the nerve fibres could be protected from auto-aggression by reconstructing the physiological electric potential by means of treatment with the neurotransmitter "2 amino ethanol phosphate" (= Phosetamin® and Calcium-EAP®). If his hypothesis is confirmed, it would appear that the electrical potential available in the healthy state of the tissue involved is essential for the ability of the B-cells to recognise substances as endogenic. Thus perhaps a certain perceptive ability, which - like an electronic key - no longer works when the battery has run down, is responsible for this recognition.

That would mean that auto-aggression could only take place if the natural electric potential has broken down¹, whilst at the same time TCR carrier complexes are also present which are bonded to endogenic structures as a replacement for their lost hapten.

In this way the origin of auto-aggression diseases can easily be understood. In laboratory diagnosis one would look predominantly for antibodies or determine antibody titres. However, the lack of haptns hinders that very production of antibodies or even makes it impossible. Thus in practice one cannot record contamination with isolated carriers using these investigation methods. As the replacement haptns favoured by the TCR carrier complexes are mainly found in the mesenchyme, they are also bonded there and are therefore unable to circulate any further. Therefore searching in the blood, which is the main substance investigated by laboratories, is futile, and it becomes clear why the causes of such illnesses have remained in the dark until now.

The nosode testing developed by Dr Voll was the first to shed some light on this darkness. The claim which he first formulated as his working hypothesis, that many chronic diseases can be explained as contamination by the very same toxins which are at the root of the nosodes found in testing to be the source material, is borne out to such a degree by the observed effects of complementary hapten therapy that it may be regarded as proven.

In this way the therapeutic combination of nosodes and haptns opens up for the first time the possibility of a truly causal

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3 Pollutants which conduct electricity or permanent electrical voltages between tooth fillings could perhaps be responsible for this breakdown, possibly also strong irritation caused by electrosmog or the influence of geopathic fields.
therapy of illnesses in the rheumatic mould, since - so far as chronic inflammations caused by T-cells are concerned - it attacks the primary causes of these conditions.

If progressive rheumatologists should ever aim for causal therapy in their specialist field, there is no getting round the use of haptenes which must frequently be combined with nosodes. Having said that, they find themselves in an almost hopeless dilemma, since up to now their treatment plan has been almost exclusively based on immuno-suppressive drugs.

Since causal treatment with haptenes and nosodes can only be effective in conjunction with a functioning immune system, it cannot be used at the same time as non-steroidal anti-rheumatic drugs, steroids or the even harsher immuno-suppressive cytostatics.

And so this solves the puzzle of the origin of inflammations caused by T-cells; the T-lymphocytes suddenly send out inflammatory mediators not because they have become crazy for no apparent reason, but because they are bonded to a toxic carrier whose destruction will be accelerated with the help of the known inflammatory reaction.

The hapten from \textit{Pseudomonas aeruginosa} works as an antigen absorber to complement the corresponding nosode \textit{Bacterium pyocyaneus} (unit dose pack F3). The hapten has a somewhat broader spectrum than the nosode, and it even seemed to me that it could be used with viral nosodes as an antigen absorber. Here however it could actually be a matter of reversing immune suppression caused by hydrocortisone. In vitro tests by Kunze and Hartmann suggest this, for not infrequently patients with chronic virus illnesses have previously been treated with steroids.

So it can happen that the \textit{Pseudomonas} hapten, e.g in the SPS (swine fever serum) nosode, unit dose pack F39, must be administered alternately with single injections of the nosode which together with a complementary remedy is required in increasing dilutions.

To be sure, assurance is given again and again that the swine fever virus cannot be passed to humans; nevertheless in numerous cases swine fever serum contamination can be found. Dr Voll reports that he himself has been through a full-blown, extremely unpleasant swine fever serum infection. For the most part contaminations like this can be explained only as non-infectious alimentary pathogenic toxicoses; they are almost always to be found in chronically inflammatory intestinal diseases, often too in stomatitis and spastic bronchitis. An overlying swine fever serum contamination must also be taken into consideration as the fundamental disease in chronic eczemas if they begin to weep.

\textbf{SANUKEHL- preparations from the company SANUM-Kehlbeck}

The company SANUM-Kehlbeck GmbH & co. KG, Hasseler Steinweg 9, D-27318 Hoyan/Germany, produces haptenes which are marketed under the trademark „SANUKEHL“. The products are offered in 1 ml ampoules and 10 ml dropper bottles n 5X and 7X potencies and are registered in several countries. All code numbers given for the unit dose packs and test packs refer to the catalogue of the Staufen-Pharma.

The following 13 hapten preparations are available:

1. \textbf{Hapten from \textit{Pseudomonas aeruginosa} (= SANUKEHL Pseu, corresponding to the Argentinian preparation Polipse)}

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\textbf{Case 7}

In 1976 G.K. - then 11 years of age - was brought to me with recurrent uveitis. My tests showed that he too required the SPS nosode. However, my control tests before each injection showed that treatment with this nosode could only be carried out successfully by administering between each hapten dose two injections of 0.1 mg \textit{Pseudomonas} hapten (Argentina), which made me think, as described above, that this hapten was also effective in virus diseases. However, the patient had previously been treated intensively with cortisone in a children’s hospital. Therefore the trials by Kunz and Hartmann named above, which give evidence that immune suppression caused by hydrocortisone can be reversed by the \textit{Pseudomonas} hapten, appeared to explain the need to give G.K. one ampoule of this hapten twice between each of the nosode injections.

2. \textbf{Hapten from bovine \textit{Mycobacterium tuberculosis} (= SANUKEHL Myc, corresponding to the Argentinian BCG hapten)}

The hapten from bovine \textit{Mycobacterium tuberculosis Typus bovinum} fits all tuberculin nosodes: Tuberculinum (= T. humanum, unit dose pack E3); Tuberculocidinum Klebs (unit dose pack E5); Tuberculavinum avis (unit dose pack E7); Tuberculocidinum bovinum (unit dose pack E8); Endometritis tuberculosa (unit dose pack K12) and tuberculosis of the bladder (unit dose pack M8).

Tuberculin contaminations are not only found in diseases of the joints, as described in Case 4 and Case 6, but can be found in practically all the organs. Even in patients with acute hearing loss, I mostly found tuberculin contamination. (See also the article about the tuberculin constitution in Sanum-Post no. 51, p.4).

As only immediate treatment has any chance of success in people affected, in my view this hapten belongs in the emergency kit.

3. \textbf{Hapten from \textit{Streptococcus haemolyticus} (= SANUKEHL Strep, corresponding to the Argentinian Estreptohapten)}

The hapten from \textit{Streptococcus haemolyticus} fits all streptococcal nosodes: Streptococcinum (unit dose pack A5), staphylo-streptococcinum (unit dose pack A28), Streptococcus viridans (unit dose pack A29),
Streptococcus haemolyticus (unit dose pack A30), Scarlatinum (unit dose pack F2) and Nosode Parulis (Streptococcus mucosus) (previously unit dose pack Z34).

In many diseases in the rheumatic mould, contamination with streptococci is involved as part of the cause. Test must be done to determine whether treatment should be carried out with one of the named nosodes. If this is the case, enough ampoules of streptococci hapten should be held ready so that if necessary drastic aggravations can be brought under control.

With all complaints which arise or persist after streptococcal infections which have been treated with antibiotics, it is necessary to consider contamination with Clostridium difficile and to think about using this hapten.

### 4. Hapten from Staphylococcus aureus (= SANUKEHL Staph)

The following nosodes fit the hapten from *Staphylococcus aureus*: Staphylococcinum (unit dose pack A4), Staphylococcus aureus (unit dose pack A26) and Staphylo-streptococcinum (unit dose pack A28),

Only the fungal nosode Trichophytie requires its own hapten (SANUKEHL Trich).

Furthermore I experienced that in the case of Mrs E.H. (Case 5) the administration of 7 amp. SANUKEHL Cand 5X immediately remedied drastic aggravation of aches and pain in the sacral region which occurred after the administration of Ustilago maydis 30X. Even if this is a one-off observation, it can be taken as a possibility that the fungal antigens of Ustilaginaceae can also be pathogenic for humans and that the carriers isolated from their antigens can also bond to the Candida albicans hapten. Contamination with Ustilaginaceae can perhaps be caused by the consumption of pork or poultry (possibly in the case of Mrs E.H. by turkey steaks), as these animals are frequently fed on maize. Corn blight is common in all maize-growing areas.

Take care if unexpected pains in the spine and other joints begin during the course of a series of injections with fungal nosodes! Not only the TCR-carrier complexes of mycobacteria and streptococci but also the fungal antigen complexes mobilised during treatment will happily attack articular cartilage.

The progress of the illness described in Case 1 might lead us to suppose that the carriers of pathogenic fungi are not only mobilised by means of a course of treatment with nosodes but also in acute fungal infections can be freed by anti-mycotic therapy. In such cases Candida hapten should be administered. Antiphlogistics will only hinder healing in this case and can turn an acute condition into a chronic one.

### 5. Hapten from Candida albans (SANUKEHL Cand)

The hapten from the yeast *Candida albans* fits the nosode Monilia albicans (unit dose pack N20) and also most other fungal nosodes, such as the nosode of mycotic fluoride (unit dose pack K18), the aspergilli: Aspergillus niger (trial pack 144), Aspergillus fumigatus (trial pack 168) and Aspergillus ochraceus (trial pack 187) - perhaps one would also consider a purge with Aflatoxin (unit dose pack A37) - Geotrichum candidum (trial pack 170), Mycosis oris (trial pack 62), Sporotrix Schenckii (trial pack 178), Malassezia furfur (trial pack 180) and Torulopsis glabratis (trial pack 146).

According to more recent findings SANUKEHL Prot has also proved its worth in the treatment of an infection with Helicobacter pylori (see the SANUKEHL article in Sanum Post No. 43, p. 2).

### 6. Hapten from Trichophyton verrucosum (= SANUKEHL Trich)

The hapten from the fungus Trichophyton verrucosum is the hapten which fits the Trichophytosis nosode (unit dose pack N14). Time and again this nosode could only be proven after preliminary treatment with the hapten, from which one might conclude that it is not a rare thing for a mixture of acute and chronic disease to apparently be able to continue over a long period in trichophytoses.

### 7. Hapten from Proteus vulgaris (= SANUKEHL Prot, corresponding to the Argentinian Proteus hapten)

The hapten from the bacterium *Proteus vulgaris* fits the Bacterium Proteus nosode (unit dose pack B2) which, as it happens, can also only be proven after preliminary treatment with this hapten. However, it must frequently be administered repeatedly after each individual dose of the nosode. Many a chronic Proteus cystitis which has resisted the efforts of specialist urologists for many years has been healed in this way. Unfortunately often 2 ampoules each of 0.1 mg of Proteus hapten were necessary for this. SANUKEHL Prot ampoules are marketed in a 7X potency and further studies still have to prove which dosages must be applied to achieve similar results.

### 8. Hapten from Propionibacterium acnes (= SANUKEHL Acne, corresponding with the Argentinian Haptenovacuna)

The hapten from the bacterium *Propionibacterium acnes* matches the nosode Corynebacterium anaerobium, which was previously sold as KUF series no. Z 49 and is now available as unit dose pack A38.

It can be used as an antigen absorber for all chronic irritations in the damp environment of the airways, and for example also for the different influenza nosodes. Naturally it can also be considered for the Acne nosode (unit dose pack N17), but could be of particular interest for the treatment of

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Acne fulminans which is not affected by any treatment with antibiotics.

9. **Hapten from Brucella melitensis**  
(= SANUKEHL Brucel, corresponding to the Argentinian Brucel hapten)

The hapten from the bacterium *Brucella melitensis* complements a treatment with the Brucella melitense nosode, Malta fever (brucellosis) (unit dose pack F34) or the brucella nosode (unit dose pack F5).

It is not necessary for someone to travel to Malta to become contaminated with *Brucella melitensis*. Brucella contaminations can apparently be acquired by consuming imported milk products, particularly from sheep’s or goats’ cheese. The mostly chronic catarrh problems must then be relieved with the corresponding nosode, whereby an intermediate dose of the hapten from *Brucella melitensis* will be required as an antigen absorber. In acute contamination with brucella, gastro-intestinal pains as in Case 5 can be the main symptom; they can be relieved quickly by an immediate dose of Brucella hapten, even without a Brucella nosode always being required afterwards.

My experiences relate here to the Argentinian ampoules, which contain 0.1 mg of Brucella hapten (Argentina). It cannot be said yet how many ampoules of the SANUKEHL preparation Brucel 6X will be needed in order to achieve comparable results.

According to more recent experiences SANUKEHL Brucel has also proved itself in the treatment of bollrosis (see also the SANUKEHL article in SANUM Post no. 43, p. 2).

10. **Hapten from Klebsiella pneumoniae**  
(= SANUKEHL Klebs)

The hapten from *Klebsiella pneumoniae* fits the Klebsiella pneumoniae nosode (trial pack 53), as well as the *Haemophilus influenzae* and *Haemophilus influenzae* serotype B (trial packs 113 and 114). Although this hapten is available in ampoules only as 6X, the required balance of toxins mobilised by the nosode can generally be achieved with three to ten ampoules.

11. **Hapten from Escherichia coli**  
(= SANUKEHL Coli, corresponding to the Argentinian Coli hapten)

The hapten from *Escherichia coli* matches the Bacterium coli nosodes (unit dose pack B1) and verotoxin-producing Escherichia (trial pack 192). In particular one should think of this hapten if problems arise with the intestine and bladder after treatment with antibiotics, but also during a course of treatment with the *Bacterium coli* nosode - as already described in Case 1 and in the section on fungal nosodes - which could be counted as of the rheumatic type. At the present time ampoules of this hapten are also only available in Europe in 7X dilution.

12. **Hapten from Salmonella enteritidis**  
(= SANUKEHL Salm)

The Bacterium Gärtner nosode (unit dose pack B50) corresponds with this hapten. As expected, this hapten from *Salmonella enteritidis* also fits the other salmonella nosodes: Typhimum nosode (unit dose pack B3), Samonella TP nosode (unit dose pack B31) and Salmonella Typhimirum nosode (trial pack 135).

This hapten may also be required after a blind course of antibiotic treatment, administered as a precaution against nosocomial (hospital-borne) infections, like the following hapten from Serratia marcescens.

Although this hapten is available in ampoules only in 6X dilution, the required balance of mobilised toxins can generally be achieved with three to ten ampoules.

13. **Hapten from Serratia marcescens**  
(= SANUKEHL Serra)

A nosode has not yet been produced from *Serratia marcescens*. In my provings the hapten from *Serratia marcescens* has occurred in the nosodes Sarcina ventriculi (trial pack 84), nosode Yersinia enterocolitica (trial pack 91) and the clostridridium nosodes: botulism nosode (unit dose pack B8), tetanus nosode (unit dose pack DA4), Clostridium difficile nosode (trial pack 21), Clostridium paraputrifucum nosode (trial pack 22), Blostridium cadaveris nosode (trial pack 123), Clostridium innocuum nosode (trial pack 124) and Clostridium tertium nosode (trial pack 125). It is to be expected that it could also be used for the Enterococcinum nosode (unit dose pack B19).

*Serratia* types are found particularly frequently in hospitalism as pathogens. It is already emerging that SANUKEHL serra is useful in very many nosocomial illnesses.

That means that in many patients who are discharged from hospitals in which antibiotics have been administered to them, with or without indications, problems could arise afterwards which refer back to residual toxices after nosocomial infections. Such problems can often be treated satisfactorily with this hapten (naturally, if applicable, also with another hapten corresponding to the contamination). This should in fact be done before such problems have deteriorated to the point where they become chronic and then require nosodes. In this age of contagious immuno-deficiency and therapeutic immune suppression, hospital doctors too ought to investigate whether the *serratia* hapten could not already be of
great importance in hospitals for the treatment of hospitalism.

The haptens from *Salmonella enteriditis*, *Serratia* and *Klebsiella pneumoniae* have not yet been made available to me from Argentina; my observations regarding these haptens are therefore not so comprehensive.

Apart from the nosodes indicated for the individual haptens, in contaminations which can be purged with a pyrogenium (unit dose packs A1, A15, A16, A18, A19, A20 and A32 and the Pyrocoxinum produced by the Archea company) all the individual haptens may be required as antigen absorbers, since when flesh decays a great variety of microbes can be involved in the process.

As stated above my experiences are based predominantly on the hapten preparations which are stored in a concentration of 0.1 mg per ml, that is in the concentration of a 4X dilution. On the grounds of theoretical law on medications, the SANUKEHL preparations may only be sold in 5X, 6X or 7X potencies. According to my experiences, often several ampoules are required in order to achieve comparable results, which on the other hand facilitates a matching of the dose and combination to the individual needs of each patient during the proving.

Possibly extensive percutaneous use of SANUKEHL preparations will also be suitable: these are available in 10 ml drop solutions in 6X dilution.

As regards the use of *Brucella* haptens as antigen absorbers in the treatment of *Brucella* contamination as described, I have also received reports that in Argentina an Interferon-inducing effect of this hapten has been observed, in particular in a combination with the hapten from *Pseudomonas* (SANUKEHL Pseu).

Meanwhile it has been proved by R. Kunze and J. Hartmann in *in vitro* tests with SANUKEHL Pseu (= Polipse) that there is a significant increase of tumour necrosis factor - 2 and Interleukin 1ß, - 6 and - 10, whereby there was also a substantial increase in the factors which stimulates granulocyte / monocyte colonies.

Such effects could also already be observed with higher homeopathic potencies, in contrast to those described above, whilst my points come from the fact that the effects of the haptens as antigen absorbers are not of a homeopathic nature but follow stoechiometric laws - that is, each free carrier molecule needs its hapten molecule.

Another preparation made from tumour cells is also available. This could perhaps be a possibility for an intermediate dose for degeneration nosodes. The few tests that I was able to do with this preparation do not however allow me to draw any conclusions: the effects in this area are certainly much harder to ascertain. It is certain that much of interest can still be expected in future research into haptens as therapy.

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