The
Behaviour of Bacteria
in the
Digestive Tract.

Allan Macfadyen, M.B. & C.M. (Ed. 1883)
M.R.C.S. Etc.
Importance of the subject.
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Researches.
A. External to the Body.
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A bacteriological subject as the ground work of a thesis, happily need not need any words of apology to commanded itself to the theoretical or practical mind. The science is like a thriving infant, which is beginning to walk a little upon its legs, and the future development of which is big with promise. But the treatment it has received has not always been judicious, and not always suitable to its tender age. The infant phenomenon has been studied by all sorts and conditions of men. Chemists, Physicists, Surgeons, Botanists, Biologists, Histologists, Pathologists, and others, inspired and uninspired; all have taken their turn. It is not to be wondered at that a reaction, if not already in progress, should be imminent. This reaction will be necessary and do good service by sweeping out those workers who are always floating on the surface of the occasion. Research will still quietly proceed avoiding the quicksand of rash theory, and building only on the solid ground of experiment, verified and verifiable.

We are at anytime aware to the fact that we have the Bacteri to thank for a large...
Part of our wellbeing and illbeing. It is easy enough to be lost amid details and to feel only the flux around us, to oscillate to Rock and Flügel, now to Hapfel and Zopf. But we can conduct our researches in a quieter spirit, and in the biological relationships of microorganisms find out much as to the endless reactions which are constantly occurring between them and their environment. Further, it is a duty demanded from medical men, as scientific watchers over the living laboratory of the human body, where according to the standpoint of today the bacteria play a role, important as that of the Prince of Denmark in Hamlet.

The behaviour of Bacteria in the digestive tract is a question of the first importance as a few words of explanation and elucidation will show. Though the smallest of organisms and lying as the confines of sight the bacteria have a far reaching influence in the economy of nature. They bring about the decomposition of dead organic matter, reduce the oxidation of otherwise resistant bodies; and execute the most varied fermentations, being in dis perseable to us w
the preparation of our ordinary foods and drinks. As parasites, they attack our cultivated plants; and induce fatal disease in the lower and higher animals, as well as in man himself. Their metabolic activity is very great, and we find the most varied products resulting, as will be seen from the following list:

- Gases e.g. Carbonic acid, Hydrogen, Marsh gas, Sulphuretted Hydrogen etc.
- Water.
- Sulphur.
- Volatile bodies e.g. Alcohol; Formic acid; Acetic acid; Butyric acid etc.
- Fixed Acids e.g. Lactic acid.
- Bases of Aromatic series e.g. Tyrocin, Phenol etc.
- Reduction products e.g. Linalol etc.
- Complicated molecules e.g. Carbohydrates, Proteins, Colouring stuffs, and alkaloidal poisonous substances.

With the commencement of the independent life of the child, air is breathed and swallowed, and with it organized germs enter the intestine, and have been found there a few hours after birth (Escherich). In the intestinal contents we find manifold processes running their course, in which
a new arrangement of elements occurs, generally also an accumulation of water, with the resultant formation of new products. These processes we call Fermentative, and when associated with the evolution of offensive odours Intestinal.

Ferments may be divided into the organized and the Soluble, and have five cardinal properties. They all belong to the organic kingdom:

1. They are active only in the presence of water.
2. The resulting products contain more Hydrogen and Oxygen than the original bodies.
3. They are capable of decomposing Hydrogen Dioxide.
4. They work best at temperatures between 30° and 35° C.

We may say that purely Physiological fermentations are brought about by the soluble ferments, while pathologicaal are due to the organized. Putting on one side the Physiological, we find a large number of so-called Pathological Fermentations.

Certain species of the (Leptog) Schizomyceles are capable of producing very different kinds of fermentations, which we find occurring in the Digestive Tract.

I. The Phenomena of Putrefaction. These consist in
a quick and intense decomposition of nitrogenuous bodies, especially the albuminaceous, by certain Schizomyceetes with the evolution of peculiar, badly smelling products in large quantity. The albuminous bodies seem first to become peptonised. As final products we find Carbonic acid; Hydrogen; Sulphhydrated Hydrogen; Sulphuric acid; Phenol; Ammonia or Propionic acid; Lactic; Linal or other Phenolines. The fatty acids formed in the process are finally through other forms of Schizomyceetes converted into Carbonic acid, Water, and Ammonia. Thus the complicated albuminous molecules are resolved into the simplest.

II. The Lactic Acid Fermentation — in which sugars as well as glycerine are converted into lactic acid. We have familiar examples in the souring of vegetables, fruit preserves, milk, and the souring of beer in part. In the human body the sugars contained in the vegetable nourishment can undergo the same change in the mouth and stomach, especially when the contents of the latter are weakly acid, or have a neutral reaction. The prime mover in this fermentation is the Bacillus acid. Lactici (Strecker)
III. The Butyric acid fermentation: — in which the bacteria convert Butyric acid out of glycine, malonate, desobutin, such sugar, starch etc. A similar process takes place in sour milk, whereby it assumes a rancid taste, and also in the ripening of cheese. The germ is most widely distributed in nature, Bacterium Butyricum (Praz mucus). It builds spores and is anaerobic. It has marked metabolic properties e.g. the cells form a ferment which dissolves cellulose and starch.

II. The Acetic acid fermentation: — in which alcohol is oxidised to acetic acid. Used practically in France for the quick formation of vinegar.

The germ is the Bacterium Aceti (Küten). All these processes can take place in the stomach, especially when the Gastric juice is insufficiently acid, or when the food remains abnormally long in the same. Remembering the role the above-mentioned germs play, we might diagrammatically represent the process thus:

\[
\begin{align*}
2(C_2H_6O) & \text{Alcohol} + 2 CO_2 \\
C_2H_6O + O & = C_2H_5O (\text{Aldehyde}) + H_2O \\
C_2H_4O + O & = C_2H_4O_2 \quad \text{(Acetic Acid)} \\
2(C_3H_6O_2) & \text{Lactic Acid} \\
C_4H_6O_2 + 2 CO_2 & + H_2O
\end{align*}
\]
V. The cellular fermentation: – associated with the development of Carbonic acid gas and Marsh gas.

\[ n(C_6H_{10}O_5) + n(H_2O) = 3n(CO_2) + 3n(C_2H_4) \]

The precise nature of the germ is not yet determined. The above is a fermentation of the vegetable Cellulose contained in the food. The industrial gases consist of, Carbonic acid, Hydrogen, Nitrogens, fuel, pured to Hydrogen, and Marsh Gas.

VI. Saracina may be mentioned: – The well-known Saracina Vorticulile (Gosford) which is found in the stomachs of healthy and unhealthy men and animals, and is abundant in cases of stomach enlargement and in Chronic Pharynx. Saracina Intestinale, (Bose) found in the intestines of fowls.

Mentsch was the first to draw particular attention to these fermentations & to bring them within the pathological field.

The writer is not aware that any serious effect on the human organism has been ascribed to the Lactic acid fermentation. On the other hand, the Butyric acid & the Putrefactive fermentations can undoubtedly produce marked effects. Further OCCURRENCE of marsh gas in proto-plasmic poison. O. Lister has demonstrated that
Butyric acid is a powerful poison acting chiefly on the nerves and brain.
Ewald had the opportunity of observing a case in which one patient expressed it as "at times the brain, at times the gas manufacturing was at work." At one time it would be the alcalie fermentation, leading to the formation of acetie acid; at another time the Butyric acid leading to the production of hydrogen and Carbonic acid. Also gas would at intervals issue from the unfortunate individual's mouth of an inflammable nature, and burning with a weak flame. This was undoubtedly marsh gas due to the additional cellulose fermentation.

Both children these processes are frequent. In these abnormal alcoholic fermentation lastly occur, accompanied by severe diarrhoea, resulting in many cases in death.

The intimate relation of micro-organisms to many disease processes being now proved, we have to remember that pathogenic germs are readily admitted into the digestive tract, and that they can find the conditions favourable for attacking, and undermining the organism. For the following examples suffice:

I. Bacillus Anthracis.
2. *Bacillus anthracis:* In Koch's words, "one can picture the life history of the anthrax bacilli in the following manner. With the excrement or blood of anthrax animals, they find a nidus on decaying vegetable structures, which in marshy neighbourhoods, and on the banks of rivers or have opportunity in the warmer months to develop, reproduce themselves, and reach the stage of spore-building. In this manner enormous spore-formers are developed resistant against the winter's cold, which settle on the margins of marshes and rivers, and in their mud. Through higher water levels and a strong flow of water they are stirred up with the mud, carried away and deposited on the flooded pasture-ground. Here they are taken up by grazing animals with the pastures, pass into the intestines and reproduce the disease."

3. *Bacillus of the myriapodae of disease, Schneirnstalbahn:* (Schütz) — These germs when swallowed reproduce the malady. In the dead animals the bacilli are found in the lungs, spleen, liver, kidney, and in the lymphatic passages.

4. *Towel Cholera:* (Pasteur). This disease can be introduced when the bacteria are added, mixed into the digestive tract. Typical
Intestinal pathological changes are found in hemorrhagic enteritis of the stools, ulceration, and therefore diarrhea. In the abscesses on the intestinal epitelium one finds the characteristic microbes.

IV. Cholera Asiatica:—In connection with this disease Koch's "singular Bacillus" may be said still to hold the field. In the intestinal canal these bacilli form the seat of their development, and biological activity. Further, they are found in the intestinal contents, in the stools, and in the intestinal wall, but not yet in the blood or organs of affected persons. In the intestine a special seat is furnished in the tissue of the tubular glands. The forms grow easily upon the normal media, as well as in blood, the human feces, and in dirty water. In ordinary drinking water they are able to prolong their life for many days.

V. Typhoid Fever:—In relation to which Gaffky has confirmed the researches of Berth, as to the constant presence of a bacillus, which is spore-bearing. It is found in the mesenteric glands, liver, spleen, and kidneys. Pfeiffer also found it in the intestinal contents and feces of patients.
during the Asiatic plague epidemic. Lastly Hesse, Fränkel and Simmonds have confirmed the above observers' statements, and have been able to produce, as they say, some of the typical typhoid pneumonia by means of the isolated bacilli. The germ would find an easy entrance into the digestive tract in the form of spores.

VI. Tuberculosis: which affects the intestine leading to ulceration. In connection with this process we have the well-known tubercle bacilli; found in all the products of animal and human tuberculosis. When introduced into the anterior chamber of the eye, or into the abdominal cavity of living animals the germs have the power of producing tuberculosis. In these bacteria we have an example of germs which can portray their entire and complete development in the living body alone. In the body they multiply, and develop in the resistant form of spores. The sputum, which is rich in bacilli and spores, may be taken as the chief source of infection. Also one can readily recognize their occurrence on the foodstuffs and in the fluids consumed, as also in the flesh of tuberculous animals. Wesley has fed animals with tubercular
material, and injected the suspension into the ileum and cæcum. In the former case the produced tubercle in the mesenteric glands, in the latter case tubercle of the intestine; mesenteric glands; and more distant organs. It concludes that by feeding with tuberculous material, only the spores preserve their virulent property, and that the bacilli through the influence of the normal gastric juice lose their virulence.

If confirmed, a notable extension of the field of pathologic organisms has lately been made by Marchiafava and Celleri, who have found in patients suffering from Malaria an organ. It is probably belonging to the class Protozoa, the Plasmodium malignant. It is a rathen hard to indicate the extraordinary interest medical men have in this branch of research, and that the behaviour of bacteria in the digestive tract demands the fullest investigation we can give.
The Researches up to the present time.

The earlier authorities considered that infection through the digestive tract did not often occur, and that the causes of disease made their way into the human organism through the lungs especially. (This idea has of late been revived by Jenner in relation to Chorea Asiatica.) A strong disinfecting power was ascribed to the gastric juice. Liebig and Becher as early as 1819 contended: "The stomach is mostly filled with food, and the cooling nature of the gastric juice weakens or inhibits the power of infectious stuffs." Indeed the digestive canal has been again brought forward as a source of infection and ascribed an important role in general disease processes.

The question remains to be answered, how is it that we take into the body so many germs without apparent injury? How is it that undressed decomposing substances, such as cheese, can be eaten with impunity, while the intravenous injection of putrid substances causes death? Here we have the fact that the causes of disease act differently according to their point of entrance into the organism. Hence the failure of action when introduced into the digestive
Tract? The most general answer has been that in the normal secretion of the digestive canal, and especially in the gastric juice, we have agents of sufficient protective power. Putting on one side the investigations which have merely an historical interest, one is compelled to say that the researches up to the present time are both insufficient and contradictory.

The authors refer only to individual forms of bacteria, and classes of animals. They often do not differentiate between spore containing and spore free material. They are often hasty as to the demonstration between the arrest of the development, and the killing of the bacteria in their experiments.

Their researches are frequently conducted with impure and mixed cultures. One notices also a failure to appreciate the distinction, or contrast, instituted to an infection of an animal with a disease germ. Further general statements are made from too limited premises, and rules are laid down, which may have been gleaned from the observation of only a few forms of bacteria. Finally the experiments are often only conducted in the laboratory, while the body presents dis.

Tissue, derivative relationships.

In conducting a Bacteriological Research it is absolutely necessary to work with pure
cultures. It is only in a pure culture that we can be sure the genus we wish to ob.
serve is really there, and is in an active state. In impure media many lose their power, are overgrown by other genera, undergo degeneration, and exhibit all the abnormalities of Involution. Thus an impure culture of Koch's Coemia Bacillus is absolutely worthless, as the Bacilli very quickly die.

Indeed, it is only in a pure culture that we can be sure we have Spore containing or Spore-free material; if we are work-
ing with the errantive genus itself, or with the extremely resistant Spores. This dis-
truction is always to be observed and stated.

When we say that a genus undergoes Arrest of development, we imply that in a given medium it is incapable of mani-
festing externally its functional activity; that this functional activity is in abeyance.
When we say that it is killed, we mean that as a living body it has ceased to exist.
In the former case the genus could still grow if removed vitreous to a suitable soil; in the latter case not at all. The wheat
grain in the land of the Egyptian mummy is in a state of arrested development, the same grain in Joseph'sSeed is dead.
To prove that a genus is killed, we must again place it under the most favourable circumstances for its growth.

The more nearly may be reached to Infection and Intoxication. To prove an Infection with a genus we must find that when the genus alone is taken, it is capable of growth inside the living body, and of reproducing the same disease from the section of which it was isolated as the cause. If these conditions are fulfilled it may be called Pathogenic.

But many germs are capable of producing marked symptoms in the body and yet are not Pathogenic. Thus we have the phenomena of elevated temperature, increased respiration, cramps, cyanosis and Collapse, which are associated with the absorption of the results of putrefaction processes. That is a pure Intoxication and not an Infection. Thus feeding pigs with quantities of cholera material will certainly produce symptoms, but these due to an Intoxication not to an Infection.

I can here best introduce a short account of some former Researches on the Subject of this Thesis, as part of a needful historical survey, and also in illustration
of the above structure I have mentioned to make.

The research of *Jack,* entitled "Infectious Stuffs in their relation to the digestive canal."

This author sets as the question to be answered:—Have the secretions of the digestive tract a specific disinfecting power over infectious stuffs, i.e. microorganisms?

He used saliva, gastric juice, bile, pancreatic extract, and putrefactive ferment (decomposing bile and decomposing pancreatic juice).

He did not think it necessary to work with pure cultures; the experiments lasted generally twenty-four hours; it conducted at the body temperature.

1. Some ordinary moulds—Penicillium glaucum and Aspergillus fumigatius. It finds that only the putrefactive ferment inhibited the mould activity; that with the digestive secretions, they could retain their vitality, so that P. glaucum could be split or disintegrated into salicylic solution into salicylic acid.

2. Anthrax material—the species of affected sheep solid in the form of pulp.

Results:

<table>
<thead>
<tr>
<th>Material</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bill</td>
<td>Retained infectious property when injected into animals.</td>
</tr>
<tr>
<td>Pancreatic juice</td>
<td>Animals survived infection. It concludes that gastric juice is disinfectant.</td>
</tr>
<tr>
<td>Gastric juice</td>
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</tr>
<tr>
<td>Putrefactive ferment</td>
<td>The same results as (6).</td>
</tr>
</tbody>
</table>
| Hydrochloric acid| Active fatal to germs above 0.1%.

13
B. Anthrax Spores.

Results: (a) Saliva
     Bill
     Panaeatic
     juice.
     (b) Gastric juice (rated, indeﬁnibly) that an actual
     act of digestion renders spores inactive.
     (c) Subcutaneous intraperitoneal results, same actual
     fermento, often spores inactive.

3. Tuberculous.

Results negative, only with active putrefactive fermento.
He found no infection through the injection
of the material into animals.

Thus come up: From these experiments it will
be seen that a comprehensive or intense dissipa-
tion process of the digestive juices or any fermento
contained in the normal digestive organs cannot
properly be spoken of.

That the power of the gastric juice where exercised is
due to the hydrochloric acid
That still enough putrefactive germs are found in
the intestines to produce the phenomena of decomposition.
Finally, that probably the digestive cannel is its
chiefest vehicle possessions as efﬁcient germifiler.

It will seem that these experiments are of a
very artiﬁcial nature. They are conducted
neither with pure cultures nor with pure media.
No indication or given to show that the Anthrax
or Tuberculous material was free from spores.
The experiments do not enable us to differentiate between developmental creep, and killing of the germs, and the proper way the cardinal question for us is does it secretion kill the micro-organisms?

Twenty-four hours as the span of an experiment does not follow by any means natural relationships.

The experiments with the putrefactive ferments are based presumably on the fact that among the products of these fermentations, we find such bodies as Phenol. And the separation of Phenol in the human body is an inconstant factor, and varies so much that we can hardly be justified in regarding it as a germ killer.

The research of Frank bearing the same title as Tachs:

1. With tuberculous material.

Small clumps filled with tuberculous tubercle, were freely divided and covered with water for twenty-four hours.

A. Therefore measured off a quantity of the fluid and set to successive quantities the following:

1. 1:1000 phenin
2. 1:2000 + 0.05-0.1% Hydrochloric acid
3. 0.05-0.1 HCl alone
4. 0.25% Bile

Incubator 37-38° C. After 1-6 hours, 5-8 cubic
centimetres of the fluid injected into the peritoneal cavity of rabbits and guineapigs. Also central rabbits injected with the pure tubercular fluid. The animals which died or were killed after six weeks showed general tuberculosis.

B. Repeated experiments

1. $1:1000$ peptic

2. $1:1000$ + $0.2\,986\,1\%$ Hydrochloric Acid

3. $0.2\,986\,1\%$ Hel.

4. $0.3\,0\,10$ Bile.

The animals also showed Tuberculosis.

2. Anthrax material - In test-brood and in part pulverized spleens of anthrax animals.

A. Sterilized distilled water 10 cm.

Blood 3-4 drops

Added to same series as under B.

The peptic fluid killed the animals within 24 hours. The bile fluid after 24 to 48 days. On the other hand hydrochloric acid fluid did not kill the Animals.

B. Repeated experiments but

1. $3:1000$ peptic

2. $1.5:1000$ + $0.0\,6\,1\%$ Hel.

3. $0.1\,2$ Hel.

4. $0.3 - 0.4\,10$ Bile.

The peptic fluid killed the animals. Defied with $0.12\,0\,10$ hydrochloric acid remained for one hour. After ten hours not do. Frank then concluded, that $0.2\,986\,1\%$ Hydro
Chlorine acid 1 per 1000 proved, even after 6 hours, is not able to destroy tubercular germs, while the autolytic material thus produced still resistant for an hour under the action of 0.125% hydrochloric acid.

Frank expresses his adherence to fresh conclusions.

What destroys the value of this research for us is the ignoring of the distinction between spore-free and spore-containing material. Did the above results apply to the bacilli, then an important addition to our knowledge would have been made, but supposing the fluid to have contained spores, we have no real advance on what we already knew.

The media of course were impure. The structure also seems to the writer as worthy of mention. Inoculations of fluid of maximum concentration are taken, as it were indiscriminately, without any reference to the body weight of the animal used, or any idea as to the enormous doses which may be accidentally given, in order to produce what should be merely an incidental effect. Frank injected 5-8 Ccm fluid into peritoneal cavity of animal. Allowing him one kilo, as the weight of his guinea pig's fasting 5 Ccm; as the amount injected, the proportion for an adult
of twenty-five cubic inches, or 6.8 litres, would be 340 cubic centimetres.

III. Professor Dr. Miller of Berlin has published two articles which require a short notice:

1. "Fermentation processes in the intestinal tract and the Schizomyctetes engaged therein."

The Professor points out that the mouth is the best filter for germs; that the tongue is the best filter for germs; and fermentation processes can be caused very easily by the smell of Stomatitis Alveolaris, of Caries; of the poisonous effects of the saliva from a finger scratched by a tooth.

As was to be expected, bacteria were found in the saliva in many bacteria and in the isolated twenty-five different forms. Of these light were found again in the stomach of a man, who could empty it at will. In the intestinal evacuations twelve were found. Professor Miller argues that the majority must be able to pass through the stomach.

To try to prove this experimentally, a mixture of bread and milk was made, so that 14 consisted of bread, of this mixture 26 c.c. put into a flask, sterilized, inoculated with a germ, and kept at the body temperature. Hydrochloric acid was added gradually till the end of two hours the mixture contained 0.2 of HCl.
Therefrom plate cultures.
He concludes that the germ is swallowed at the beginning of a meal and passes through the stomach, but that in two to three hours only the very resistant are able to do so.

This second research is entitled "Some germ forming Schizomyces of the digestive tract, their fate in the stomach, and their reaction on different foods."
These germs he had found to be very resistant to artificial gastric juice, to eat. With this for the animal body arranged a series of dog experiments.
The animals were fed with a mixture of meat, bread, milk, and sugar, beside 10 cubic centimetres of a mixture of four of these germs. In 24-26 hours all the dog had diarrhoea
1st. Killed in 2 1/2 hours after last meal.
2nd. " 6 " " " " " "
3rd. " 8 " " " " "
4th. " 9 " " " " "

Also a small loop pulled out of stomach content of No. 1 gave 1,000 colonies on the plates. From No. 4 none living.
He concludes that germ can live 6-8 hours in stomach of healthy dog. These researches however do not settle
the question. The distinction as to shore or not drawn of a large quantity of food is given. In short, the mixed diet, and the symptoms produced, remove the experiments somewhat from the normal conditions or at any rate do not give the gastric juice a fair chance.

The mixed food also would contain multitudes of germs, which would closely resemble those described by zeller, and which must, as a matter of fact, be already widely distributed in nature.

Thus zeller, frank, and zeller, while agreeing in their conclusions, base the same on experiments which are inadequate and not final.
The present state of our knowledge.

I. The Gastric juice.

It has been distinctly proved that some forms of bacteria are killed by the gastric juice, whilst others are uninjured. The comma Bacillus of Koch—

He says:—The comma bacilli are killed in the stomach, for when onefeed on

meat with cholera fluids, or with cul
ture, and then feed them after some

time, no comma bacilli are found

in the stomach, or in the intestine. This

abstute Koch sought at first to over

come by injecting into the duodenum,

as avoiding the stomach. Finally

he sought to overcome it in another way

Taking Carbonate of soda (50th) solution,

he introduced 5 Com. into the stomach

of the guinea-pig for three hours the stomach

gave an alkaline reaction.

Seven guinea-pigs were killed after

twenty hours. In one, cholera bacilli

were found in small intestine, show

that they had passed through the

stomach, but without producing illn

sue. It occurred to him that the absence of path

ological effects might be due to the in

testinal Peristalsis. He then injected
opium into the abdominal cavity (5 c.c.m. for each 25 gm. body weight of the animal). Rectum was narcotized last up 1 1/2-2 1/2 hours. Finally, neutralising the gastric juice, and injecting opium, the animals were fed with cholera bacillus 35 became ill, and 30 died with cholera symptoms.

Experiments made in this fashion succeeded also with the ‘Weidler and Prior’, the ‘Buch’ and the Miller Shindlew.

Also succeeded with anthrax bacilli (which are killed in sheep’s stomach), Cholera de Poules, and the rickettsia of rabbits, the animals being handled as above.

With regard to spores, Koch found a resistance against the action of the gastric juice. He fed guinea pigs with spore containing anthrax bacilli.

Results:

1. After 2 hours: Many spores in stomach, in cecum already a few.

2. After 3 hours: Larger number of spores in stomach, many in small intestine and in cecum.

3. After 3 1/2 hours: Few in stomach, still many in intestine and in cecum.

4. After 5 hours: Very few in stomach & intestine, very many in cecum.
These experiments, as will be seen, were made with vegetable eaters whose digestive phenomena differ much from those of flesh eaters. Thus currently killed and peps the stomach is forced, stuffed with a fine mass of food, so that it is more introduced and it difficult to pass through. Further, the small intestine, in comparison with the stomach, is found almost empty. The stomach is strongly acid, the small intestinal alkaline: the large esophagus has contents distinctly acid. As food when introduced does not get properly mixed up in the stomach, as in flesh eaters, but forces its way through, in the order in which consumed. Finally, very through the stomach, the food mass passes quickly through the small intestine into the caecum.

For many other reasons, inadequate research, it remains doubtful if they can pass through the stomach. On the contrary, micrococci and Spore free bac. tend. Dr. Stadek has the statement that only Spore building bacteria are able to do so, but experimental proof of this same is lacking.
II The Bill.

As to the Bill and its action, we find no concensus of opinion, the most contradictory statements being made as to its antiseptic action.

In giving a synopsis of the literature we make a natural start with the classical researches of Bedder-Schmidt. As their statements have been the key note for much that has more recently been written, it may prove interesting to quote their own words:

In days with bile jaundice they noted the following phenomena in the bile-free intestine. 

1. The defecation seemed to go on in the proper manner, the intestinal emptyings were sluggish and rare, the faeces soon assumed a greasy, clayey property, were grey or greenish coloured and displayed an extremely bad after

pungently foul smell, which distinctly pointed to putrefaction. This was also indicated by the strong gas development, the constant noises and rumblings, and the giving off of badly smelling flatulence. The smell of the inspired air was un

pleasant, equally if animal was past

ing or had eaten . . . without

violent phenomena followed a
decline of the vital forces, a general illness;
mus, which after some time brought on
death. In animals, which received a
large quantity of nourishment — which
for the deficiency of fat were made more
resistant through much Albumen and
Carbohydrates, the phenomenea were modi-
"fied, but the gas development in the
intestine and the character of the faeces
remained the same.

Although these decompositions occu-
only with animal-diet, viz. with
more exclusively meat-diet, were the
animals fed daily with bread, Bar.
"sorzyme and Flatus were a matter of
fact occurred in an unusual degree,
but the faeces and gases were almost
without smell, and the former e.
"miled an acid reaction, such as
intact animals with the same diet
seldom presented. The acid ferment
"lation, which with vegetable food
regularly occurs in the intestinal
"causal, appeared here, through the
"defective influence of the bile, these
"mounted ones the ordinary standard.

On agreement with these observations Yale
writes: "In the process of digestion the
albuninuous bodies consumed are changed into mobile peptones, while a part decomposes contemporaneously. The albuninuous putrefactive products are inferior to the or. gaseous, under their influence it becomes, and the final result of the chronic pox, being is the pathological death.

The bile acids, one may be allowed to think, act connectively towards decomposition as antiputrefactive bodies, without it is true completely preventing these processes. When the bile flows out through the fistula putrefaction becomes rapid. The result is diarrhœas and earlier death without local disease.

The bile at ends are disinfecting agents which work almost through the entire length of the intestine.

3. Schirrmann was led to think that the putrefactive process could not only be studied by observing the intestine and feces, but also by noting the absorbed products found in the urine.

In the summer of 1881 he established fistulae in two dogs, and gave for several weeks meat and a form of gruel in sufficient quantity.
Results.—In putrefaction, most dogs reared
maintained like normal animals. The
faeces firm and no trace of diarrhoea.
These unexpected results raised two ques-
tions i. Was the bile really an antisepticisation
i.e. after its exclusion from the digestive
tract are the putrefactive processes increased?
ii. Do Bidder's chemical researches find their
explanation in its hindered fat absorption?

If, on side of intestine no abnormal phen-
omena appeared, then it must be possible
to preserve a dog, after establishing a fistula,
and with equivalent diet, in the same state
of nourishment, especially the same body
weight, as before the fistula. It must also
be noted if the peculiar up of nitrogenous
constituents of the food is the same as
before the fistula. Also the Fat must be
estimated, and it must be seen if its
pores through unchanged, or if free
fatty acids are formed, and in what
quantity. Yet the days be fed, before and
after the fistula, with equal nourishment.
Compare amount of sulphuric stercor and
aromatic organic acids discharged in the urine,
measure as these are dependent on the
intensity of intestinal putrefaction.

Results.—In fed with meat extract, results
had place, the corresponding colour of the
food. A flatulence. The constipation and smell not manifestly changed. The feed up with a moderate quantity of fat free homestyle the same results.

Misused diet:—it a putrefactive phenomena. With meats and quantity of fat:—faeces became gradually grey brown, grey yellow, finally grey, till they become diarrhoeic. This diarrhoea disappeared as soon as the cause; the fat, was removed.

Conclusions:—Intestinal instincts of absence of bile is still active enough to uphold badly functions— that it is in an abnormal condition where a quantity of unabsorbed fat is present—that cause of intestinal phenomena is not due to anti-putrefactive failure of the bile, as is proved by estimation of offensive substances in urine. (Feces acids volatilized in C.02 not increased in days with bile pastules) By equivalent nourishment, just as much nitrogen absorbed from intestine as before. Also days can retain the same body weight.

4. Researches of Ideal and Fruits:—

Fruits absorb a versatile acid in part in its antiseptic action to the Bile acid, especially the Tannosalic acid, which last he fruits as an antiseptic in many respects
little beneath tannic acid and the phenol.

It must direct his attention to the cause of putrefactive processes, water-organizing and using antiseptic in dogs sense of the word, that enough is established in order to prove the bile acids antiseptics, if we find that they are able to hinder the development of putrefactive germs.

I. Influence on putrefactive and fermentative phenomena:

A. Beef + water. 38-40°C. In 24 hours bacterial growth.
In 2-3 days cloudy and intense stink.

Instead of water Bile acid solution (or part suspension) then used, only given concentration above phenomena no longer appeared. A few flakes, kept some time in dilute saturated 'bile acid', can be washed weeks long in pure water at incubation temperature, without any appearance of cloudiness, smell, or bacterial.

B. 20 C.C. 10% Cholic acid solution; Glysocholic acid solution (suspension); in increasing quantities + 0.5 gram meat (finely divided beef) 3 days in incubator. Then contents of each glass microscopically examined.

Following results:
<table>
<thead>
<tr>
<th>Concentration</th>
<th>Description</th>
<th>Bacteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.0%</td>
<td>Turbid; no smell; no mould;</td>
<td>No Bacteria.</td>
</tr>
<tr>
<td>0.5%</td>
<td>Turbid; no smell;</td>
<td>No Bacteria.</td>
</tr>
<tr>
<td>0.2%</td>
<td>Turbid; a few moulds</td>
<td>Round-germs.</td>
</tr>
<tr>
<td>0.1%</td>
<td>Turbid; almost no smell; odour; many moulds</td>
<td>Round-germs.</td>
</tr>
<tr>
<td>0.05%</td>
<td>Turbid; sticky; many moulds</td>
<td>Round-germs.</td>
</tr>
<tr>
<td>1.0%</td>
<td>Glyceraldehyde: clear; no smell;</td>
<td>No Bacteria.</td>
</tr>
<tr>
<td>0.5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.2%</td>
<td></td>
<td>No rods; some moving cells.</td>
</tr>
<tr>
<td>0.1%</td>
<td>Turbid; faint stick; Rods and cells.</td>
<td>Many moulds.</td>
</tr>
<tr>
<td>0.05%</td>
<td>Turbid; stomp stick;</td>
<td>Many moulds.</td>
</tr>
</tbody>
</table>

From above experiments, it is concluded that glyceraldehyde or succinicolacetic acid (0.2%) hinders...
the development of Pseudomonas fluorescens, which he considers the characteristic Schizomyces for putrid meat water. He considers that the appearance of moulds in Glycocholic acid solution (1 p.c.) does not impair his results, notwithstanding that there was no excess maturity. The Glycocholic acid was only potent against some forms, while Taurocholic acid had a more general antiseptic action. Under similar conditions, active concentrations of Salicylic acid and Phenol were respectively, 0.1% and 0.2%.

B. Experiments with Pancreas:—
  Taurocholic acid, 0.5%, arrested development of putrefaction, while 2% of Glycocholic acid was not enough, the process being only delayed.

C. Wine decomposition:—
  Glycocholic + Taurocholic Acids, 1%. First putrefaction signs in 2-3 weeks.
  0.1% was not sufficient.

The most recent researches are those of Bufalini and Lindberger.

5. Bufalini's experiments:—Like much be investigated the action of the Biliary
on principles upon the putrefactive process.
The action of animal principles on pancreas
putrefaction, & that of blood serum,-
the reaction cancels the presence or absence of
Bacteria microscopically.

A: 10 (10 gr.) pancreas infusion. In 24 hours 
(1) $10 \text{ cc. water salina of } \chi$ chalic $²$
(2) $10 \text{ cc. water salina of } \chi$ chalic $²$
(3) $10 \text{ cc. water salina of } \chi$ chalic $²$
(4) $10 \text{ cc. water salina of } \chi$ chalic $²$

B: 5 (5 gr.) blood serum + yeast, 
(1) $3 \text{ cc. } \chi$ chalic acid $²$
(2) $3 \text{ cc. } \chi$ chalic acid $²$
(3) $3 \text{ cc. } \chi$ chalic acid $²$
(4) $3 \text{ cc. } \chi$ chalic acid $²$

B. (1) 5 c.c. blood serum + yeast. 
Qs development 9/2 c.c. 
(1) 3 c.c. + 2 c.c. 3 c.c. 
(2) \text{ chalic acid (0.7\%)} 
(3) \text{ chalic acid (0.35\%)}

But alcohol places \text{ glycochalic acid first, and then
chalic acid second, in antiseptic power.}
It will be seen that while he holds as doxin
stated, that the \text{ bile acids hinder putrefactive
processes, the hindrance is not a complete one.

6 Lindbergen Research:—
Lindbergen canniderin is necessary not only to investi-
gate the action of the bile acids, but also of other
acids which cause to present in intestinal
Canal, e.g. hydrochloric, lactie, o acetic acids.
Also the working of the bile, by alkaline or by neutral reaction. As experimental material he used water, pancreas infusion.

The entrance of putrefactive phenomena was in part estimated by microscopic observation of development of bacteria in the fluid, and in part by the severe smell. Temperature employed was 40° C.

He found that alkaline fluids became quickly putrid, and much more quickly than the very weak acid or the neutral. The presence of acids even in small quantity could completely prevent putrefaction; and 0.01% hydrochloric acid was enough to make putrefaction during several days completely impossible.

But low degrees of acidity one could not wholly prevent the entrance of bacteria in the fluids, while putrefactive smell was ever present.

By accordingly fluid curd, stoechiometric the entrance of putrefactive bacteria was hindered by 0.2%.

With 0.05 - 0.2%, after five days only a few Bacteria, while the fluid had an acid; not a stinking smell.

Lactic acid acted much weaker than the others. With less than 0.005% the fluid behaved like the neutral test.
The experiments with Bill or with Bill choids were conducted in part with neutral and in part with acid reaction. In the latter case the fluid was first brought to the desired acidity, and then mixed with 0.5 gram boiled mucous free, or with 5 c.c. fresh bullock's bile, per 100.

He found that bile by neutral reaction had no antiputrefactive power, while in the acid fluid a strong arrest of putrefaction was manifested.

With lactate choid, 0.05 00% alone almost without influence, but 0.005 00% in presence of bile, manifestly delayed putrefaction.

After a fortnight's digestion such a test contained a few bacteria, but it had a bile-like smell, and was only faintly suggestive of putrefaction.

The neutral bile containing tests examined with Bacteria and had a stinking smell, with capricious gas development.

To sum up:—Bider and Schmidt, and fully, as the result of clinical observations on daps with Bill jaundice, held that the antiseptic action of the bile must apply to putrefaction but been established.

Kühnlein adapting the same methods comes to entirely opposite conclusion, which are
further confirmed by his careful chemical analyses. He finds in the nature of the diet, and especially in the abnormal presence of unabsoled fat in the intestine, the true cause of the antirectal phenomena. That his contention is cogent and is lead to believe, especially when remembering the phenomena of retusosis; where, in the absence of bile from the intestine, the increased fat in the faeces causes them to smell most offensively.

Smith advances still further in method and seeks to reinvestigate the subject in the light of our present knowledge. He uses the individual patent caustic mixture of the bile, and investigates their action on saprophytic bacteria, and draws the distinction between the arrest of the development, and the death of the germs. He finds a marked arrest of development to occur, which is due chiefly to the action of the Faurocholic Acid

Poupart holds that the antireptic action is but partial, and is more marked in the case of the Glycocholic acid. Lindberger finally finds the Antireptic power to be very strong, but to be only increased when the bile is acid, this
acidity being presumably due to the bile acids set free by the acids of the Chyme.

It will be seen that:

Their results are contradictory.

They are based in great part on merely unscientific observation, and the cause of error.

The cultures used are impure.

They refer only to putrefactive processes.

No contrast is made between the bile acids and their salts.

They give only partial conclusions, from the limited number of germs observed.

### III Some other Factors.

Several observers have found that in the intestinal caecal more bacteria are to be distinguished microscopically than can be recognised again in culture, made from the contents of the same, outside the body. Such observed by means of the microscope in the intestinal contents of healthy men the greatest variety of bacterial forms, I considerably extending the number he was able to recultivate outside their host.

This is in contradiction to the statement of Renstok, that the number of forms
found is few. Meanwhile such re.
searches leave it undecided whether we
have to do with an additional factor un-
related to the bacteria or not. I mean
if one has in these cases to do with
injured and enfeebled Bacteria;
jured through the acids of the food
juice, the intestinal secretions, or ferments,
or with bacteria which are not recultiv.
able on the usual culture media with the
ordinary methods.

The whole subject required reexaminations
and while painfully conscious of the
length and breadth of the theme, I venture now
to detail the researches I was able to make as
to the Behaviour of Bacteria in the Digestive
Tract.
The experiments fall under two natural
groups, intended to be complementary,
the one to the other.
I. Experiments conducted outside the body.
II. Experiments conducted within the body.
I. Experiments conducted outside the body.

In these experiments the attempt has been made to follow out certain principles. I elected to make the researches with a variety of bacteria, pathogenic and non-pathogenic, and of varying susceptibility, so that the results might be of general application, and correspond more to the state of matters in the body, where we have thevigilance of all forms of germs.

It was also sought to copy where possible natural conditions.

Further, I selected germs which could readily recognise again.

A distinction was made between spore-bearing and spore-free bacteria. I made sure that all the cultures were pure in these respects.

I worked only with pure cultures of individual bacteria, obtained by independent cultivation.

Also I distinguished carefully between the arrest of the development, and the killing of the bacteria.

It would be tedious to relate here all that pertains to the technique of the laboratory work—such as sterilisation of apparatus, material
the method of preparation of media; and technique: suffice it to say that the most approved methods of Koch, under the personal supervision of Professor Carl Stieff, were followed here.

Germicide material:-

- Ureum: Chloride 1 per 1000; Absolute Alcohol, 10% heat, up to 180°C.

Cultivating media: -

Gelatine and Agar. Agar—(Neutral)

<table>
<thead>
<tr>
<th>Gelatine</th>
<th>Peptone</th>
<th>Salt</th>
</tr>
</thead>
<tbody>
<tr>
<td>5%</td>
<td>7%</td>
<td>10%</td>
</tr>
</tbody>
</table>

Agar 1% 

Other fluids for general use:—

- Sterilised distilled water: 2% sterilised salt solution (0.85%)

- Colouring agent:— methyl blue, alkaline solution, renewed every fortnight.

Ever glass microscopic preparations made by allowing a portion of the object to be examined to dry on the glass, after having been conveyed there with a sterilised platinum wire. Then passed three times lightly through the flame of a Bunsen Burner. Finally cleaned and soaked in distilled water, briefly and solution of Mounier's medium was Canada Balsam.
The microscope used was a Zeissel (Sættungen) with homogenious oil immersion (114) —

List of Bacteria employed in form of pure Cultures: — It does not belong to this Thesis to give an account of the morphology of the following forms. Each has well-marked characteristics, soon learned, & facts which enable one to distinguish them as required. I had no difficulty in picking them out of their differentiating appearance from one another —

1. Staphylocoecus pyocyaneus Aureus.
   (Koenenb. Mikroskopische bei den Kranken. Lethargiekrankheiten. Lpz. 1884.)
3. Leprospheo Bacillus. Isolated this from one of the catarrhagues meat infusion. Short bacilli arranged generally in rows. Lyophiles gelatin with development of green colour & characteristic redish stain. On plates lyophiles gelatin in 3-4 days, at 15° C.
4. Bacillus Pyocyaneus - gramine Ester (Gersaud. de la pyocyane et de la ménètre. 1882)
5. Microcoecus Tetragonus.
   (Koch J. Gazett. 1877. 7. Bd. 2. Abth. 4.)
6. leptococcus of Rabbits. These bacilli colour more strongly at the poles than in the middle.
7. Anthrax Bacilli. Spore free by culturing very slowly at a temperature not exceeding 14°C, and not under 12°C.

8. Bacilli of Typhoid Fever. (Spore free). Cultivated at 15°C.


10. Finckh's Pneum Bacillus - Cholera bacillus.

The investigations.

1. The Gastric juice.

The medium employed was gelatine. At first I used gelatine tube cultures, to which had been added the factors of the gastric juice, in the hope that I abandoned this i in these researches and in all subsequent experiments made use of gelatine plates, for the following reasons:—when one takes the material as a platinum wire, and inoculates from gelatine, thereon, one can readily inquire that the organism is not under the full influence of the medium.
The platinum needle forms a sequestration in the gelatine, in which the organisms grow. The colonies at the side are perhaps killed or suspended by the medium, but they in their turn could form a wall within which the remainder grows. In this case, in a measure, develop. In the case of gelatine plates, the germs are added to the liquefied gelatine, thoroughly mixed up, and the whole poured out on the glass plate. On stiffening, the isolated colonies are caught up again in different portions of the gelatine, and remain under the intimate action of the medium.

An important procedure was the case of temporary marking of control plates. The germs in identical quantities were taken, reintroduced into the same amount of pure gelatine, and the whole also poured out on glass plates. Thus, for purposes of comparison, we had side by side, a natural growth of the germ, and its growth as determined in the experimental medium.

Method of preparing the germs:

Heredised distilled water - 10 c.c. intr.
Divided into a sterilised test tube. If fresh, pure culture of the organism taken, or enough of the same added to the water, till
a cloudy mixture was obtained. Then a sterilized glass pipette (which was made and required) was filled with this bacterial fluid, and two drops of the same into dried into 5 c.c. sterile distilled water in another tube and the same plugged with cottonwool. From this two drops were again taken and introduced into the various gela
tive media experimented with, as well as into the culture gelatine. Well shaken up and finally poured out on the glass plates.

In the result of experience I found that this gave me a well tracable plate, and I was able to feel that the germs were not aggregated in clumps, but were fairly divided up in the mixture.

This method was used in all cases.

Subsequent experiments made with gastric juice, Bull etc.

Pepsin — I was able to heat it up to 100°C. It so sterilize when dry with.

but its losing its properties. It was dried in a desiccator.

Hydrochloric acid — Specific gravity 1.1982. Taken (containing 40.4 III) and diluted. Its strength then determined by titration with bismuth nitrate.

The above then added to the gelatine in the necessary quantity. The concentration
always made on 100 c.c. gelatine at least.

The gastric juice: —
1. Pepsin 0.3 %
2. Hydrochloric Acid ... 0.2 %
3. Mucus.
4. Mineral salts ... 0.2 %

The experiments A. Pepsin alone.
B. Hydrochloric acid alone
C. Pepsin + Hydrochloric acid.

A. Pepsin

The mixture with gelatine emulsolated with ten representative germs. Control plates also made, according to method described above.

The query: — Has the pepsin itself any action on micro-organisms — or if so what? Does it arrest the development of the germs? If not, it will vitiate them —

<table>
<thead>
<tr>
<th>Temperature 15° C.</th>
<th>0.2 %</th>
<th>0.5 %</th>
<th>1 %</th>
<th>2 %</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Staphylococcus</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2. Vibrio Parahaemolyticus</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>3. Aquasol Aerogenes</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>4. Antovar Ranae</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5. Hulstner &amp; Porc</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>6. Leptodermus Auratus</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>
The experiments were made three times.

Conclusion:—No hindrance of development by the pepton when acting alone, and, a fortunate check-up of the bacteria.

B. Hydrochloric Acid.

Method:—Here I began to simulate the natural state of streaks. I questioned importance in, how the hydrochloric acid killed the germs? As the experiments were limited to the time of an average digestive act. (four hours). They were conducted at the body temperature (37°C). After this lapse of time other temperature I sought to find if the germs were dead or, not, i.e., capable of growing again when removed to suitable soil.

Gelatine + Hydrochloric Acid 0.05 – 0.5 vol.

Two drops of the bacterial fluid added to each of the various concentrations. Then in the incubator at 37°C. for four hours. At the end of this time a quantity was moved with sterilized pipette from each of the tubes, and of this, one drop, and five drops, introduced into pure gelatine, to give a concentrated and dilute mix. Transferred mixed up, then focused on plates, and kept under observation at 15°C.
also the control experiments conducted in exactly the same manner. The control plates would likewise indicate of the bac.
love which I had used were really in a vital and normal condition.

The Query: Is the hydrochloric acid the active antiseptic factor in the gastric juice, as stated by many observers, and if so does it act in natural quantities to powerfully as to kill the bacteria which find their way into the stomach, or prevent their passage into the intestine?

The experiments tabulated underneath were repeated five times. The boundary was taken as lying between the last plate to show any colonies and the next one above it which had none. The diluted and Concentrated plates only showed a difference in the number of colonies, and did not make any difference as to the bounded areas indicated in the table. The + sign indicates where any colonies were to be seen, the 0 sign indicates empty plates. All the control plates developed in normal fashion, and were full of colonies. They were all examined with a much magnifying power of the microscope. Here are the results shortly tabulated:
### Hydrochloric Acid

<table>
<thead>
<tr>
<th>Method described above</th>
<th>0.05%</th>
<th>0.1%</th>
<th>0.2%</th>
<th>0.3%</th>
<th>0.4%</th>
<th>0.5%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Staphyl. Aureus.</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2. Typhus Bacilli.</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3. Micrococcus Tetrag.</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4. Anthrax Bacilli.</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>5. Septicemia of rabbit</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>6. Koch's Comma Bac.</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>7. Fricke's Prior.</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>8. Sarsophytes Bacill.</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>9. M. Protoggres.</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

### Conclusions:

The hydrochloric acid is the active germicide in the gastric juice, but the action is much less powerful than had hitherto been supposed. We find certain germs very resistant against the action of the mineral acid, though placed under circumstances the most favourable for its action. Thus we find that under an imititation of natural conditions and concentrations, certain germs are still able to survive its action. Its precise boundary can be fixed, but each germ must be examined on its merits.

Staphylococcus Aureus and Typhoid Bacilli are very resistant to proportions higher than we would expect to find in the normal juice. Also Micrococcus Tetragone, a Potrefactive Bacilli resistant.
in the other hand Anthrac. Bacilli and Ruber Septicaemia are susceptible; while Koch's Bacilla bacilli, the Streptoc. Prior, and the Micrococci prodigiosi are most susceptible.

At present, we cannot imagine that in the body many germs would pass undifficultly from the stomach into the intestine in a healthy state—while others would undoubtedly be killed.

At any rate these experiments do demonstrate that the power ascribed to Hydrochloric acid in not borne out in fact; in short that it is limited.

C. Pepsin + Hydrochloric Acid.

The Query:—Does the pepsin then make a difference, by lowering or by raising the boundary of the action of Hydrochloric acid. Does the fermentative action pres. serve the genus for a constant inactivating action of the Hydrochloric acid; or by dissol. the cellulosic membranes.

For this experiment I selected Typhus Bacilli (Aspergillus), and Micrococci Tetrafomes.

Peptolysine action was tested first, and the amount of pepsin taken enough to ensure the same vig. a peptolysine
of the gelatine medium. The experiments conducted as before B., and control plates were made.

If the peptic aided the hydrochloric acid then one would find the boundary between growth and failure of growth lowered. If it retarded the action of the acid then the boundary should become higher.

---

<table>
<thead>
<tr>
<th>Hydrochloric Acid + Peptic</th>
<th>0.1 %</th>
<th>0.0 %</th>
<th>0.7 %</th>
<th>0.4 %</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typhoid Bacillus</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>Repeated 14 times</td>
</tr>
<tr>
<td>Moraxella Catarrhalis</td>
<td>+</td>
<td>+</td>
<td>0</td>
<td>0</td>
<td>Twice</td>
</tr>
</tbody>
</table>

Contrast plates full of bacterial colonies easily identified as those of the respective germs.

Conclusions:—The boundary limit of action remains the same as in series B. and the peptic does not help, and does not hinder the action of the hydrochloric acid.

---

Does the amount of peptic make a difference in these respects?

The same methods followed as to above four concentrations of hydrochloric acid. The following proportions were respectively added of Peptic 0.1 %

\[ \begin{array}{c}
0.1 \\
0.3 \\
0.6 \\
1 \\
\end{array} \]
The writers do not feel called upon to undertake a full consideration of the spores and of numerous organisms. Hydrochloric acid, a watery 2 per cent. solution, only killed certain spores on the tenth day.

Further, experiments of Nash already done failed, i.e., often have demonstrated that spores can easily withstand the action of the gastric juice.

To crow up these results:

The finding:

1. Peptic alone does in no way hinder the development of the germ, but still less kills them.

ii. The hydrochloric acid is the active factor in the gastric juice, but its influence is not such as the laws of previous research would lead us to expect.

iii. That these experiments, conducted outside the body, cannot lead us to conclude that...
the acid places a serious barrier in the way of germs, in their passage through the stomach to the intestine.

4. That while it is true that several germs are very easily killed, such as Koch's Canine Bacillus, Rabbit septicaemia, and Shille's animal, representative pathogenic germs, such as Typhoid Bacillus, are very resistant, as also a typical germ of cutaneous ulceration; the saprophytic Bacilli.

5. That the resistance of certain of the germs is against such a strength of acid that a priori we might say that they could pass, a stomach containing a normal proportion of hydrochloric acid, without being killed, with much greater facility in states of lessened acidity.

6. That the addition of pepsin and the consequent peptonicizing action of the pepsin on the microcide does not aid or retard the acid in its work, so as to make any perceptible alteration of the boundary towards a lower or a higher limit.

7. The quantity of pepsin added does not make any difference.

8. That spores are very resistant as a matter of fact survive action of gastric juices.
II. The Bile.

1. What action has the bile itself?
2. What action have its patent constituents, i.e., the bile acids?
3. What action have the bile salts, and what contrast do they present in their action to the bile acids?
4. What action have the bile acids in the presence of other acids?
5. Will one be justified in concluding for a strong antiseptic action unone or any case?

It will be sufficient to show that there is an arrest of development, in order to prove the role the bile is assigned in the digestive tract. The following experiments are then arranged to investigate gases, as arrested in their development.

A. Bile alone.

Frequency:—Has it an antiseptic action?

Fresh pig's bile was used both of neutral and nearly alkaline reaction. It was sterilised and mixed with 7 per cent gelatine in proportions of 20/10, 5/10, and 10/4. Inoculated with gases, according to usual method, and then from plates, to also control gelatine plates. Temperature
of 15° C., when control plates had grown, the bide plates were examined.

Following organisms tested:

1. Staphylococcus Aureus
2. Typhoid Bacilli
3. Micrococcus Tetragonus
4. Anthrac Bacilli
5. Aeroplastic Bacilli
6. Koch's Casma Bacilli
7. Smolens & Priorn
8. Micococcus Prodigiosus

Results: All grew as well as on the control plates, even with addition of 1% per cent of bile.

Conclusion: The bile itself, in alkaline or neutral state, in no way arrests the development of micro-organisms.

B. Bile Acid.

Glycocholic and Lanthocholic Acids.

Query: Are they the antiepticies of the power ascended to them, and in the name of the word that Carabolic acid is? Do they have a potent action on pathogenic and non-pathogeneous germs, or at least a potent control over putrefactive processes, so that an important role can be assigned to them for one or other respect, in the intestinal canal?
I sought here not to use pure factitious germs merely but to have a variety, pathogenic and non-pathogenic, and varying in nature and in susceptibility. I introduced two new germs into the experiments:—

1. Pasteur Bulgaricus (Hauser, Iver Jædelius. Bacterin. Impf. Leipizig 1885) This is a psychrotrophic Pasteur Bulgaricus which rapidly liquefies the gelatin and produces a nespectum acide. Hauser has assigned it a rôle in septicaemic processes.

2. Immunerth Bacillus — (Immerich, Archiv fur Hygiene, Volume III. 1885) Immunerth believes it to be the germ of Cholera Asiatica, but the culture I used was taken from Cæcum of a rabbit, in no way suspected from Cholera. I also isolated a germ having identical properties, out of the Stomach of a dog. The germ is reported to be very resistant.

In these researches we must try to preserve natural conditions, I not so experiment with amounts of the acid which could hardly be present in the intestine at one time.

It will be useful to try to form a mental picture of what occurs in the intestine. What influence has the acid. Stomach
Cheyne au ile bile when discharged into the duodenum. We must imagine that the bile acid salts cease to exist, when they enter the intestine, and that there we have the free acids. As so far as the acids of the stomach enzyme are stronger than the bile acids, in so far will the least be set free through the first, or at least our equipoise will be established alongside of the presence of free bile acids. At the same time no acid is destroyed or disappears, but only hydrochloric acid (or lactic acid) are replaced by the equivalent bile acids; or one may say the titrination acid quantity remains equal.

I may say that so far as intestinal contents react acid, so far must free bile acids be present—so in the intestine we can only think of free bile acids being present, their neutral salts no more.

In Human Bile Glycocholic is much in excess of Deoxycholic acid. In the dog only Deoxycholic Acid is present.

Deoxycholic Acid: \(-\text{C}_26\text{H}_{45}\text{NO}_7\).

Glycocholic Acid: \(-\text{C}_26\text{H}_{43}\text{NO}_6\).
Analysis of organic constituents of Human Bile. - (Hoppe-Seyler).

Water

Water

Organic matter 91.68

Organic

Nucleus 1.29

Bile salts (Taurocholate)

Taurochrome 0.87

Glycocholate

of Taurine 3.03

Soaps

1.39

Cholesterine

0.35

Lechithin

0.53

Fat

0.73

I obtained taurine acid from physiol.
real chemistry laboratory in Leipzig.

Taurochrome acid was unsoluble
in water, but with glycocholic acid
secured any amount as strongest con.
centration, 0.5 0%. Beyond that part
in suspension. I only used the latter
in proportions in which it was soluble.

The neutral solutions of serums
were made, and the same quantities therefrom
filtered. Also control plates. Neutral gelatin
was impregnated with the acids & salts to
given concentrations (I may add that in
the experiments the mixtures were made
with neutral gelatin). Temperature 15 ° C.
Series I. Taurocholic Acid.

(The data given is for the experimental conditions described in the text. The experiments were repeated 3–4 times.)

<table>
<thead>
<tr>
<th>Taurocholic Acid</th>
<th>0.2 %</th>
<th>0.5 %</th>
<th>1 %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Saprophytic Bacilli</td>
<td>No D.A.</td>
<td>No D.A.</td>
<td>No D.A.</td>
</tr>
<tr>
<td>2. Proteus Vulgaris</td>
<td>No D.A.</td>
<td>No D.A.</td>
<td>No D.A.</td>
</tr>
<tr>
<td>3. Green Pus Bacillus</td>
<td>No D.A.</td>
<td>No D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>4. Immuniczki Bacillus</td>
<td>No D.A.</td>
<td>No D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>5. Typhoid Bacillus</td>
<td>D.A.</td>
<td>D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>6. M. Tetraploides</td>
<td>D.A.</td>
<td></td>
<td>D.A.</td>
</tr>
<tr>
<td>7. Koch’s Comma Bac.</td>
<td>D.A.</td>
<td></td>
<td>D.A.</td>
</tr>
</tbody>
</table>

D.A. = arrest of development.

Series II. Glycocholic Acid.

<table>
<thead>
<tr>
<th>Glycocholic Acid</th>
<th>0.2 %</th>
<th>0.5–6 %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Saprophytic Bacilli</td>
<td>No D.A.</td>
<td>No D.A.</td>
</tr>
<tr>
<td>2. Proteus Vulgaris</td>
<td>No D.A.</td>
<td>No D.A.</td>
</tr>
<tr>
<td>3. Green Pus</td>
<td>No D.A.</td>
<td>Partial D.A.</td>
</tr>
<tr>
<td>4. Immuniczki Bacillus</td>
<td>No D.A.</td>
<td>No D.A.</td>
</tr>
<tr>
<td>5. Typhoid Bacillus</td>
<td>No D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>6. M. Tetraploides</td>
<td>D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>7. Koch’s Comma Bac.</td>
<td>D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>8. Strephyl. Adenon.</td>
<td>No D.A.</td>
<td>D.A.</td>
</tr>
</tbody>
</table>

D.A. = arrest of development.
Series 3. Continued experiments for these genes where in previous experiments the upper and lower limits of developmental arrest had not been reached.

<table>
<thead>
<tr>
<th>Taurocholic Acid</th>
<th>0.05%</th>
<th>0.1%</th>
<th>2.0%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Leptospira bacilli</td>
<td>No D.A.</td>
<td>No D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>2. Pasteur fusacesi</td>
<td>No D.A.</td>
<td>No D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>3. Typhus bacilli</td>
<td>No D.A.</td>
<td>No D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>4. M. tetragonus</td>
<td>No D.A.</td>
<td>D.A.</td>
<td></td>
</tr>
<tr>
<td>6. Hapt. Aerobes</td>
<td>No D.A.</td>
<td>D.A.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Glycocholic Acid</th>
<th>0.1%</th>
<th>0.2%</th>
</tr>
</thead>
<tbody>
<tr>
<td>M. tetragonus</td>
<td>No D.A.</td>
<td>D.A.</td>
</tr>
<tr>
<td>Koch’s Comm. Bac.</td>
<td>No D.A.</td>
<td>D.A.</td>
</tr>
</tbody>
</table>

Remarks:-
As to Series 1. It must be at once noted that a striking and important contrast manifests itself here. The contrast between the action of the taurocholic upon the protosplastic and the pathogenic genes. The protosplastic displayed a surprising resistance to the acid, while the pathogenic became more readily to the same.

In the case of the Leptospira bacilli and the
Proteus vulgaris there was no arrest of development. With 0.5% lactic acid, while even 1% only delayed the appearance of the germs two days. The pseudomonas bacilli were arrested by 0.5%.

The Salmonella typhosa proved itself resistant, 1% being required for the arrest of its growth. With 0.2% the developmental arrest was complete. Typhoid bacilli, N. tetragonon, Koch's comma bacillus, and staphylococcus aureus.

Series 2. The same distinction manifests itself with lactic acid, though the effect is less potent than with lauric acid, as we see with 0.2% typhoid bacilli are still able to grow as well as staphylococcus aureus. The pseudomonas bacilli also suffer no arrest with 0.5% of the acid. The salmonella bacilli again proves itself resistant. The most susceptible are the pseudo comma bacillus.

Series 3. It required the strong concentration of 2.0% of lauric acid to arrest the development of the putrefactive bacillus, and the Proteus vulgaris. For the pathogenic bacilli, the arrest may be said to cease between 0.05-0.1% of...
Haemochalic acid

and glycochalic acid the arrest of development of the more susceptible *M. Tetraonurus* and *Comme Bacillus* ceased between 0.1 - 0.2%.

C. Bile Salts.

Employed haemochalate of soda, which is very soluble, and sample the usual methods, as already described.

<table>
<thead>
<tr>
<th>Haemochalate of Soda</th>
<th>0.2%</th>
<th>0.5%</th>
<th>1%</th>
<th>2%</th>
<th>5%</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Staphylococcus</em></td>
<td>NoDA</td>
<td>NoDA</td>
<td>NoDA</td>
<td>NoDA</td>
<td>D.A</td>
</tr>
<tr>
<td><em>Protuber Vulgans</em></td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>D.A</td>
</tr>
<tr>
<td><em>Strept. Pus.</em></td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>D.A</td>
</tr>
<tr>
<td><em>Immunistic Bacillus</em></td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>D.A</td>
</tr>
<tr>
<td><em>Bac. Typhoid F.</em></td>
<td>&quot;</td>
<td>&quot;</td>
<td>D.A</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td><em>M. Tetraonurus</em></td>
<td>&quot;</td>
<td>&quot;</td>
<td>D.A</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td><em>Comme Bacillus</em></td>
<td>&quot;</td>
<td>&quot;</td>
<td>D.A</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td><em>Staph. Aureus</em></td>
<td>&quot;</td>
<td>&quot;</td>
<td>D.A</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
</tbody>
</table>

Remarks: This fact practically does not arrest the development of pathogenic organisms, while its influence on the pathogenic is but a mild one. With 0.2% no arrest of development. First traced its action with 0.5%, but at times acted only reveal itself in the concentration of 5%.
To sum up these series of researches:—

Have the bile acids a potent disinfectant action? We find that the development of pathogenic germs can be arrested by \( \text{sodium} \)-cholic acid in the dilution of 0.1 \% and by \( \text{glycocholic} \)-acid, 0.2 \% solution. But it required 0.5 \% of \( \text{sodium} \)-cholic acid to arrest the putrefactive process. The \( \text{glycocholic} \)-acid is of much weaker power. The \( \text{sodium} \)-cholic acid is the potent factor.

The Bile salts do not practically come into the question.

These experiments reverse the relationship usually held as established, namely, as the control exercised by the bile acids is not over the processes of putrefaction but rather over the genus of a pathogenic nature; if, i.e., if it can be called a control. Thus the one cannot ascribe to the bile acids the role assigned to them by B. and H. with respect to the phenomena of putrefaction.

We cannot call their disinfectant power to react with alkaline and carbolic acids.

D. Bile in presence of other acids.

The Query:—does the Bile when acid in reaction exhibit a marked bacteriolytic
power. Are Leidhberger's results to be accepted as proving this?

Acids were selected which could be present in the intestinal canal of lactic acid, butyric acid. Fresh neutral bile was used also. and the concentrations were made in a 10% gelatine. Proportions were taken which could be expected in the intestinal canal and which, if Leidhberger's results were correct, would confirm his views.

Thus mixture = \[ \text{gelatine } 10\% \]

The acids were taken in pro. Bile 5.0/60

See table for specific weights. The acid 0.05 0/60

Also representative figures taken.

<table>
<thead>
<tr>
<th>Temperature (°C)</th>
<th>Acetic Acid 0.05 0/60</th>
<th>Butyric Acid 0.05 0/60</th>
<th>Lactic Acid 0.05 0/60</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Lactophyloic acid</td>
<td>No D.A.</td>
<td>No D.A.</td>
<td>No D.A.</td>
</tr>
<tr>
<td>2. Green R. Bacteria</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>3. Typhoid E. Bacteria</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>4. L. Polysaccharum</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>5. Campylobacter</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
</tbody>
</table>

Results: - An arrest of development in the bile of acid reaction. Indeed the bacteria grew as well, if not better on the bile plates as on the control plates.
Hydrochloric acid 0.05% + 3% Bile + in 10% gelatin gave identical results.

According to Lindheimer, if one found, for example, that the acid alone, in proportion of 0.1% per cent, arrested the development of the glands, and a smaller quantity 0.05% in presence of bile would have thesame effect, presumably

I suppose by setting free the still more

potent bile acids. But these experiments

of I have made, and those later, to be
detailed show that this is not the

Case. The boundary with the organic

acids was not crossed.

Conclusion: — The bile in the presence

of the organic acids in proportion which

according to Lindheimer, should be present

does not arrest the development of the

Salmonella Bacteria. or of the pathogen

genus.

A further query arose: — Is the boundary

then raised higher when bile is set to

the organic acids? i.e. are the germs

enabled to resist a though concen-
tration of the acids? 

The experiments again conducted

in usual manner. at Temperature of

15°C. Proportions of Acid 0.1 and 0.2%
may state that these results are the same as those obtained with the same organic acids, without addition of bile, as well be seen from the last series of experiments.

<table>
<thead>
<tr>
<th></th>
<th>Acetic acid</th>
<th>Butyric acid</th>
<th>Lactic acid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.1% 0.2%</td>
<td>0.1% 0.2%</td>
<td>0.1% 0.2%</td>
</tr>
<tr>
<td>1. Leptotrichia bacilli</td>
<td>D.A D.A</td>
<td>D.A D.A</td>
<td>D.O D.A D.A</td>
</tr>
<tr>
<td>2. Staphylococcus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Typhoid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Pseudomonas</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Leptotrichia bacilli</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Conclusion:— The bile makes no difference with the percentage amount of the organic acids required to arrest the development of bacteria, in the way of raising the limits above those found potent for these acids acting alone. The series of experiments with the organic acids under the heading "other factors" will show that Ziedler's results are in all probability to be explained by the powerful action of the organic acids he used, even in neutral reaction. So turn up—

1. The bile itself plays no role, either in alkaline or in neutral reaction, as an
antiseptic in the intestinal canal.
2. The Bile salts also have no power.
3. The Bile acids are not disinfectants, as many and much have stated, of such power as to warrant the conclusion that they have a strong control over the phenomena of putrefaction, while the formic acid is a potent factor to effect is only moderate, it is increased, not in the putrefactive, but in the more purely pathogenic organisms.
4. The addition of the organic acids to the Bile makes no difference in the phenomena observed.
5. The potency of the organic acids themselves is sufficient to account for the phenomena observed, on their addition to the Bile.

III. Some other Factors.

A. The acids of the Chyme.
The query—Do the organic acids of the digestive tract play any rôle in arresting the development of bacilli and further if so does that action explain Lindheimer's results when he added them to the neutral or alkaline Bile?
Again the usual methods were adopted. Temperature 15°C. The acids were added to 5 per cent gelatine, in per centages according to weight, viz. acetic acid, butyric acid, and butyric acid.

The results obtained, with these acids in the proportions of 0.5% and 1% respectively, shall not be tabulated, as in these proportions the acids arrested completely the growth of the bacteria.

The succeeding experiments were made with strengths 0.05% - 0.2%.

<table>
<thead>
<tr>
<th></th>
<th>Acetic acid</th>
<th>Butyric acid</th>
<th>Lactic acid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.05%</td>
<td>0.1%</td>
<td>0.2%</td>
</tr>
<tr>
<td>Lactobacillus</td>
<td>No DA</td>
<td>D A</td>
<td>D A</td>
</tr>
<tr>
<td>Green Piece</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Typhoid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wild Tetrapod</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shigella</td>
<td>No DA</td>
<td>D A</td>
<td>D A</td>
</tr>
</tbody>
</table>

Conclusions: These organic acids have a potent effect in arresting development of putrid, putrescent and pathogenic bacteria. Until they are the most potent factors that have found. They can be assigned an important role in the digestive tract in the way of arresting the development of the most virulent forms of bacteria. The lactic acid is of weaker power than the others.
Yarber, the protective action of these acids is due, perhaps, to explain Luedeking's results when he added them to the bile.

The above results led me to examine a little the literature of this subject and I found two theses, which confirmed the experiments above detailed.

Hoffman, investigated Formic acid but as it has poisonous effects I dismiss it with the remark that he found it to work strongly against putrefaction.

That also confirms his observations to putrefactive phenomena. The effect on them of organic acids - butyric, lactic, oiticic, lactic, acetic, citric, acids on the concentrations were made vegetative as nutrient substrates, but in pure cultures were used, putrid meat infusion, swarming with bacteria, being added to the experimental medium.

But lactic acid, that found that 0.1% & 0.2% caused delay putrefactive phenomena till the fifth day.

0.1% Butyric acid delayed putrefactive till the eleventh day.

0.1% lactic acid also delayed phenomena till the thirteenth day.

0.05 Formic acid delayed till the eight day.
It will be seen that these results support the statement the writer ventured to make that the organic acids have strong anti-pestic properties.

B. The Pancreatic Juice.

That this alkaline fluid has no influence hardly requires to be stated, but has only to remember the selection of Pancreatic infusion for experiments with Petrol facies, on account of its rapid and easy decomposition.

C. Intestinal Ferments.

The phenomena which led me to examine them were the following:—

I found a constant microscopical difference between the ordinary intestinal contents, and the mucous flakey of the same and of the intestinal wall, while the intestinal contents showed microscopically many and varied forms of bacteria, the flakey only presented a few and of limited kind. I found this in the intestine of the rabbit, dog, and man. Thus for example I could isolate under microscope, in the intestinal contents, perhaps ten different kinds of Bacteria, in the mucous flakey...
only one or two. In the flasks often limited to one third of the entire volume.

The genus also in these flasks seemed small and not much developed.

Further, these flasks on plates liquefied the gelatine very quickly round about them, if any were on the same plate, the whole of the gelatine would liquefy and run off the plate, but without smell or phenomena of putrefaction. In fact, the gelatine, as we might say, was peptonized.

The gelatine liquefied round about the flasks, generally contained no bacteria. Also on these plates there was a great increase in the growth of colonies, without they did grow, they were often moulds. Finally, some of the bacteria seen in the flasks could not be re-cultivated on the usual media.

The query: Do these intestinal flasks contain active ferments which act injuriously on the Bacillaria—hence their paucity; their fineness; and their absence in the immediate neighborhood of the flasks on the gelatine plate. In short do the Bacillaria which are visible, especially visible, which yet fail to grow in the media, fail to do so because they are either killed or at any rate enfeebled by the ferments.
Can we try assigning a disreputable role to such intestinal ferments?
I took flasks from small intestines of rabbit, dog, & man & put in small sterilised glass dishes. Then mixed thoroughly with a culture of the coli
saccharosus Tetragonus. Also a second series with Typhoid Seros bacillus—
Also control plates. Portions of the mixture of flasks of genus were introduced into gelatin, observed & also on plates.
Both genus grew, but in slightly dimin-
ished numbers, as compared with
the Control Plates of the same.

After many experiments, I was not able
to find any definite effect resulting,
beside the increased number of bacteria.
Conclusion:—No definite conclusion
can be come to, which would war-
rant us to assign any role to these
intestinal ferments. If they have any
action it would be any that of slight
weakening the Bacteria.
These experiments conducted outside the body lead us to conclude:
1. That the gastric juice cannot be taken as affording us any efficient protection against the entrance of bacteria into the digestive tract.
2. That the bile acids also in this respect have a limited action, which with respect to putrefactive phenomena is extraordinarily weak.
3. That the intestinal ferments cannot be reckoned as potent.
4. That the organic acids of the food enzyme are antiseptic, and have been found to be the most potent factors in the digestive tract, having an action that is intense and variable.

II. Experiments conducted within the body.

I have already explained the objection to using vegetable eating animals. I selected for my experiments healthy dogs, as the objection is more related to that of man. They also have a particularly active gastric juice containing much more free hydrochloric acid than the human. Average acidity in dog is
0.3 per cent.

The idea was to arrange the circumstances so as to give the gastric juice the best possible chance of exerting an antiseptic action. Then to feed the animals with microorganisms and to search for them again in the intestines. This would enable one to answer the question, does the gastric juice kill bacteria in the living body? From the results to argue back to the human gastric juice.

For these reasons I used dogs; produced only a slight filling of the stomach, by feeding with a small quantity of food; especially with small quantity of lean meat. Starved them for 24 hours, so as to get clear animal vital; and a concentrated gastric juice. Also after feeding, during digestion of food, they allowed them no water, so that the ferments might not pass too quickly through the stomach. After 4-5 hours the animals were killed by inhalation of chloroform—under the influence of which they died in a few minutes. Instantaneous death was also produced in two by passing a curved knife through the foramen into base of brain, in neighborhood of medulla. This was done to remove any suspicion to the results obtained in the dogs killed by means of chloroform.
The section was at once made, and with sterilised instruments. All the surfaces were also cleansed with Bioklone of Mercury (1 per 1000).

The following parts were double-destroyed and removed:
1. Stomach.
2. Duodenum - middle portion 4 inches.
4. Ileum - upper part 4 inches.
5. Ileum - lower part 4 inches.

These were placed in sterilised dishes with glass covers, opened at once with sterilised instruments, and contents removed into smaller dishes. When necessary, some sterilised distilled water added, but not more than a few cubic centimetres.

By means of platinum loops portions of contents taken and introdureed into the culture media: Gelatine 4 0/6 or 10 0/6 agar 1 0/6

From these attempts attenuated cultures were made, in a series of gelatine tubes of agar tubes. Finally poured out on plates kept at temperature of 15°C in case of gelatine; at 35°C in case of agar cultures.

Germ material taken from pure cultures,
spore free. I used only genus I was well acquainted with, and which could easily be recognised again in the plate cultures. Also a series of genus used of varied nature.

First experiments were very primitive. Dogs were fed much jellies, ifacces, etc., examined, but putrefaction developed too quickly, and faeces were too inter
mittent to allow of any clear results.

First I fed two dogs with milk (200 c.c. respectively) + 1/10th of sultrase Ba.

silli and microbaces. Tetragonus. They were killed after five hours. I found no sulfurase Bacterii in the intestine, and only two colonies of M. Tetragonus in lower part of ileum. This impressed one with the rapid transit that takes place when fluid is given - and I resolved to take smaller quantities, and solid food mostly.

A. Series of Three Experiments with Staphylococci. Three Mice.

Dogs were starved 24 hours. Then the
food + the genus given (to each con.


levels of 1/4 after tube cultures of Staph. Aureus).

Drinking two waters taken away. The mice were killed at the end of 5 hours - 4 plates from each portion of

intestine.
The numbers refer to the numbers of colonies found on the four plates.

Staphylo-coccus Aureus

<table>
<thead>
<tr>
<th></th>
<th>Day I</th>
<th>Day II</th>
<th>Day III</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>100 c.c. bean meal killed after 5 hours</td>
<td>100 c.c. meat killed after 5 hours</td>
<td>100 c.c. killed after 5 hours</td>
</tr>
<tr>
<td>Stomach</td>
<td>90 colonies</td>
<td>189</td>
<td>2</td>
</tr>
<tr>
<td>Duodenum</td>
<td>None</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>Jejunum</td>
<td>12</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Ileum, upper portion</td>
<td>150</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Ileum, lower portion</td>
<td>None</td>
<td>11</td>
<td>700</td>
</tr>
<tr>
<td>Large intestine</td>
<td>None</td>
<td>300</td>
<td>Countless</td>
</tr>
<tr>
<td>Stomach, anterior</td>
<td>Acid</td>
<td>Weakly acid</td>
<td>Acid</td>
</tr>
</tbody>
</table>

Observations: These results are very striking. The stomach is placed under circumstances the most favourable for its action, and yet instead of finding none of the cocci in the intestine, we find numbers i.e. they have passed through the stomach without having been killed, having proved themselves capable of re-cultivation again outside the body. Even in the stomach we find a number, which have survived during five hours there.
It will be noted that since through the slow
wash, they have passed very quietly to lower
part of the gut. When bland diet (rich)
was used the transit was very rapid
and staphylococci were found in countless
members in first portion of the large
intestine.

It will be noted that this coincides with
experiments carried out in the body, where
the staphylococci, survived the action
of hydrochloric acid (0.3%) during
four hours.

Conclusion: - The gastric juice has placed
no obstacle in the way of the passage
of these germs in a living state into the
intestine.

[Note: This experiment was arranged to get rid
of a possible objection.]

I was resolved in subsequent experiments
to strengthen if possible still more the action
of the stomach i.e. to give any meat, and
in smaller quantity and to observe more
the time of a natural act of digestion, killing
the daps after four hours. Finally to exclude
large intestine. 3 to observe only small intestine
as the important field for us.
B. Experiment.

This experiment was undertaken to get rid of a possible objection. It might be said that by starving the dogs, I had lowered their vitality and that the gastric juice was as a consequence weakened and abnormal.

The germ used was Staphylococcus aureus.

The dog was starved for 24 hours, and then fed for 48 hours with 1000 gram meat during the two days or was as follows:

1st day 12 midday 125 gram.
6 p.m. 125 "
10 p.m. 125 "
500 gram.

2nd day 7 a.m. 125 "
12 midday 125 "
6 p.m. 125 "
10 p.m. 125 "
500 gram.

3rd day 7 a.m. 125 "

Then water taken away.

12 midday 50 gram lean meat.

Staphy: aureus (4 cultures)

4 p.m. Killed.

Results: Staphylococcus aureus, Dog IV

<table>
<thead>
<tr>
<th>1. Stomach</th>
<th>20 Colonies</th>
<th>The reaction of the stomach was strongly acid. Cons.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Duodenum</td>
<td>90</td>
<td>-</td>
</tr>
<tr>
<td>3. Jejunum</td>
<td>40</td>
<td>Vanished no solid remains of food.</td>
</tr>
<tr>
<td>4. Jejunum (upper part)</td>
<td>6</td>
<td>-</td>
</tr>
<tr>
<td>5. Jejunum (lower part)</td>
<td>40 Colonies</td>
<td>-</td>
</tr>
</tbody>
</table>
Remarks:— The Coccii still were able to pass through the stomach alvei, under these conditions so favourable to the antiseptic working of the gastric juice.

Series C.

This series was arranged in order to test a variety of organisms, and of varying susceptibility, as will be seen in the tables of action of HCl, under the heading "gastric juice." The germs used were:

1. *Campillus Bacillus* of Koch—Contents of the tubes—A very susceptible germ.
2. *Epticaerum of Rabbit*—4 fresh cultures. This germ also delicate.
4. *Marbon Bacillus* (Antirax) and free from spores—VII per cent. Rabbit inculcated with antirax & after death, the liver & spleen were taken & inoculated with some meat & given to the dog to eat. They were taken immediately after death and were free from spores as usual. Copious inoculation of a portion showed.

Also control plates were made from a portion of the spleen, and antirax colonies grew, showing that the organs were in a vital condition.
The days were warmed for 24 hours. They
fed with 50 grains wheat the germoprod
after four hours were killed.

Tables of these experiments:

<table>
<thead>
<tr>
<th></th>
<th>Day V</th>
<th>Day VI</th>
<th>Day VII</th>
<th>Day VIII</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Commissary</td>
<td>Rabbit lettuce</td>
<td>H. Pseudotox.</td>
<td>Catarrhe Bacilli</td>
</tr>
<tr>
<td>Stomach</td>
<td>0 Colonies</td>
<td>None</td>
<td>5 Colonies</td>
<td>None</td>
</tr>
<tr>
<td>Intestines</td>
<td>None</td>
<td>None</td>
<td>4 &quot;</td>
<td>None</td>
</tr>
<tr>
<td>Jejunum</td>
<td>4 Colonies</td>
<td>4 &quot;</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Lower part</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Lower part</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

To prove in case of Rabbit leptomycosis that Colonies found in Jejunum were really those of that organism, a mouse was inoculated from them. In 12 hours it died, and in blood of heart and in spleen the characteristic Bacilli were found.

Remarks:— These results are of great interest.

Only two of the four genera passed the stomach alive viz. Listeria, Bacillus & Rabbit leptomycosis. The genera which were killed in the stomach were the Commissary Bacilli and the
Anthras Bacilli. The last two are very delicate. The stomach then only proved a pro-
etector again Anthras and Bacilli Comma
Bacilli, and that under the most favourable
conditions for the action of the gas tro-fusil
which it received to the writer to devise,
while the Rabbit denfereum, a delicate
germ, was able to reach the intestines
alive. These results comprise the experiments
conducted outside the body.
Thus we have found germs passing
through the stomach alive, and reaching
as far as the ileum and beginning of
the large intestine.

Series D.
Method and idea:—To take the germs
I had failed to kill with my Cholera and
Bacilli, i.e., which had been killed in
the stomach and to arrange the experi-
ments with them in a different form.
In fact to make the conditions those
least favourable to any injury of the
Bacteria viz. a quick passage through
the stomach, and that by means of a
purely fluid medium.
Accordingly two days starved for 24
hours. Then to each 200 c.c. water
given, uninfected with Cholera and
Antitoxin Bacilli respectively - 2 cultures of each. Then no further, and the animals killed after 4 hours.

<table>
<thead>
<tr>
<th></th>
<th>Day IX</th>
<th>Day X</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Antitoxin Bac.</td>
<td>Cancer Bac.</td>
</tr>
<tr>
<td>Stomach</td>
<td>4 colonies</td>
<td>None</td>
</tr>
<tr>
<td>Intestines</td>
<td>1 one</td>
<td>1 one</td>
</tr>
<tr>
<td>Lymphatics</td>
<td>3 colonies</td>
<td>None</td>
</tr>
<tr>
<td>Spleen (upper)</td>
<td>1 one</td>
<td>None</td>
</tr>
<tr>
<td>Spleen (lower)</td>
<td>1 one</td>
<td>None</td>
</tr>
<tr>
<td>Stomach React.</td>
<td>Acid</td>
<td>Acid</td>
</tr>
</tbody>
</table>

Remarks: - Thus we find that gastric fluid and a consequent quick passage into intestine, a form so delicate as Antitoxin Bacillus can pass through a normal stomach. This minimizes still further the protective value of the stomach as against the incursions of Bacteria. Of all the forms of varying nature & susceptibility I only failed with Cancer Bacillus of Koch, one must remember how delicate it is, how concentrated & strong the gastric juice of the dog it was - one can readily imagine that with a weaker gastric juice much fluid enter Cancer bacilli could pass into.
the intestine.

These animal experiments, which may be considered as complementary to the artificial researches, confirm the latter and justify the conclusion that we have no special protection afforded us in the gastric juice. Further, given the weaker human gastric juice, with mixed solid and liquid diet, pathogenic and nonpathogenic germs will constantly be passed on alive into the intestine. Also abnormal states, with lessened acidity, will still more favour the transit.

Finally, the germs once in the intestine will find, as I have demonstrated, no formidable foes in the intestinal secretions or fermento. Thus the perils there will be the organic acids of the food.

Some concluding and complementary researches:

I. Do germs pass through the normal intestinal wall?

Do the germs find their way from intestine into organs and blood, and vice versa? I was prepared to investigate
this more minutely but relinquished the research, as it would have overstepped a research of Dr. Coyness-Hawdon's working in this laboratory.

Bearing in mind the passages of fat globules, one is led to the idea that the genera in the gut might also find their way through the intestinal wall.

The center fed a dog with a large number of *Staphylocoecic Murei*, and then killed the animal after eight hours. Gelatin plates were made, but no *Staphylocoecic Murei* were found in blood, mesenteric glands, liver, spleen, Dr. Coyness-Hawdon's, with *Bacillus Leducii* and *Bacillus Subtilis*, also had negative results.

Indeed one does not feel called upon to consider such a migration as likely having regard to epithelial lining and connective walls of the gut.

The writer believes that a migration can only occur when blood passes over from gut or comes into gut, as for example in haemorrhage. The genera can migrate with the blood, i.e. only when a solution of continuity is present.

Ribbert has stated that when microscopic...
favored a wandering of bacteria through the healthy epithelium, in the follicles of the processes vermiformes of habits. In the deeper layers of the intestinal wall, in mesentery glands he could recognize no bacteria and he concludes that they are there killed. He believes, contrary to Meticshikoff, that an intra-cellular digestion of the germs takes place — Bizzozero has lately come to the same conclusion, but his researches are as yet defective.

We can readily picture a constant battle as going on between the lining cells of the body and the micro-organisms, in which fight the lining cells usually gain. In fact Meticshikoff asserts that the leucocytes of the blood are special enemies of the bacteria and that they destroy them.

The bacteria, in the normal living tissues, find mechanical hindrances to growth and a pre-accumulation of disposable nutrient. We may well imagine that the living tissue, having become potent against the bacteria than the morbid leucocytes, are as assumed by Meticshikoff.

In fact with intact epithelium and
The compact intestinal wall we have an efficient germ filter furnished.

II. Are there germs which microscopically we recognize in intestinal contents and which are not capable of recultivation, killed or suspected Bacilli, or are they germs which do not grow quickly because the usual media and methods of research are not favourable for them? Can one increase the number of germs recultivable, by other methods? In short the question is are these germs Anaerobic? This is quite a priori possible; we can imagine the anaerobic process beginning even in the mouth. Between gums and teeth the air is shut off, when the lips are closed and during sleep, and the anaerobic germs could develop. They do not require a continual shutting off of air to favour their growth. When lips closed a during sleep the anaerobic germs could develop - when air present they temporarily cease developing, while the aerobic grow - and so on.

Further we have distinctively Anaerobic forms present in the digestive tract e.g. the Clostridium Butyricum, and the Bacilli.
of malignant ulcers. The latter found in intestine of rabbit, and is exclusively an
anaerobe. Also there are germs which while
most active in presence of oxygen, still
grow without air, though flower of
Bacillus Saprophytes. (Roseoback).
As the writer made the following inves-
tigations to see if, after cultivation germs
without access of oxygen, many grew
and if there remained an important
surplus still of unattainable germs
after doing this.

**Method:** Gelatine 5 o/o
Sugar 2 o/o
Agar 1 o/o
Sugar 2 o/o

Look respectively a dropfull of intest-
inal contents of small intestine of
dog and of rabbit, put in sterilised
glass dish, a added to the same 20 drops
of sterilised distilled water and sterilised
well up. From this, with a sterilised
fine pipette, one drop:

1. On cover glass, for microscopic examination.
2. In ordinary gelatine for cultivation by the
ordinary methods.
3. In super-gelatine or agar for cultivation
with inclusion of air.

This could establish a comparison.
From a microscopic picture of all the genera contained in this portion of intestinal contents, from 1 or 2 of all the genera which would grow using the ordinary methods, from 0 of the anaerobic genera present in the same.

This method would enable us to conclude for all the genera, and to come to some definite conclusion.

Use for anaerobic experiments round glass dishes with margins 2 centimetres high. These vessels filled at least 1 3/4 centimetres deep with the gelatine or agar, as many of the genera are delicately anaerobic e.g., helicobacter sedens is exclusively anaerobic and grows best at depth of 1 centimetre.

Gelatine at 15° C., agar at 35° C. Apparatus: usual hydrogen gasator (iron, sulphuric acid, and a drop of platinum chloride) - gas conducted through lead acetate solution finally through solution of pyrogallol in potassium.

Anaerobic apparatus. One does not need to describe in detail. Stout copper circular vessel, lined with grease, and mixed with tightly fitting lid having
two apertures furnished with stopcocks. Led by means of cork and fastened down on a broad india-rubber braid. Before use tested in air tight by means of a manometric manometer.

The culture were placed in perforated metal trays inside the apparatus, & lid then renewed down. & then passed through apparatus for half an hour. Then stopcocks closed, & whole placed in incubator for 4-6 days, then opened and plates examined. A apparatus a culture of Motiers Bulgaric placed to absorb any oxygen still present.

Remarks It would lengthen an already long thesis to give an account of the morphology of the Anaerobic genus found. Besides the morphology of the genus is a detail which hardly comes within the writer's province. Let the following remarks suffice. The writer was able to isolate five distinct anaerobic genus out of the digestive tract which were quite different in the growth of their colonies & their microscopic appearance.

In the plates, there were in those from the rabbit very many fastening grown, but the anaerobic plates from days…
were quite full.
The microscopical picture presented the usual variety of germs. Thus one, presented in the field, ten different forms of bacilli. Of these four were recontrollable on the ordinary media, and three as anaerobic, leaving three forms over which had not grown.
The four forms of anaerobic isolated: 1. Fine long bacilli, presenting in their appearance and in their cultures all the characteristics of Maleplana cholera bacilli.
2. Thicker and larger bacilli—about same size as those of cholera.
3. Bacilli rounded at ends and colouring best at poles, like the bacilli of Cholera de Paules.
4. Short Bacilli with rounded ends—usually arranged two and two.
5. Small cocci in tiers.

Remarks:—I was thus enabled by cultivating with air exclusion, to get a number of germs to grow, which did not grow at all which were wholly absent from the plates treated in the ordinary manner.

Conclusion:—Quite a number of intestinal germs are anaerobic, and many of those which are microscopically seen must be
active, and not sequestrated bacteria, lacking only a proper nutritive medium. The germs which are still not accounted for, may be considered to be bacteria sequestrated through the gastric juice, the organic acids of the food, and perhaps the intestinal secretions.

Morphology

The morphology of the bacteria of the intestine not falling within province of this research it will be sufficient to mention chief researches on the subject.


His assertion that only certain species of bacteria can pass through stomach can be held as disproved by the researches detailed in this Thesis.

2. Eberleich. "Die Darminbakterien des Neugeborenen und Säuglings" | Fortschrift d. med. No 16 and 17 (1885).

Remarks as to digestion—

A point worthy to note is the phenomena presented in stomachs of cows of the dag experimented with. Even after a strong defensive act lasting during four hours an important secretion (not recorded or written in the tables) of solid food was found in the stomach, indicating that the digestive process was by no means completed within that time.

When water is consumed a portion must pass very quickly through the stomach into the intestine, as is proved by the finding of pathogenic Bacilli in the small intestine when given mixed with water. In the case of milk, being careful, it can remain probably much longer. As to milk digestion Kühnemann found that when simple milk was given it left stomach after 4 hours. When it was boiled it left after 3 hours, and when alkaline in two hours. Thus the alkaline media could easily destroy Bacilli very quickly into intestine.

As to hydrochloric acid in gastric juice: Ewald found:—
1. Preliminary stage, 10-20 minutes in which lactic acid present in stomach.
2. Stage in which as much free lactic acid as hydrochloric acid.
2. Stage in which only free hydrochloric acid is present. 30-60 minutes.

We find a weakened gastric juice in disease, anaemia, gastric colic, nervous states, motor disturbances, and a delay of food in stomach, & consequent fermentations. (Lactic & butyric, & gases developed.) In stomach ulcerae, as a result of action of bacteria digestive phenomena may easily pass into the putrefaction. The absorption of such products has probably much to do with the cause of obscure general bodily symptoms.

Concluding Remarks

The researches the writer has made show that in the whole a slender pro. action against Bracton is afforded to us through the secretions of the Digestive Tract, and especially under natural conditions; a great mass of nutritive, meaty, much fluid, with easier gastric disturbances, & consequent lesserened acidity.

There must be a frequent passage through of Bracton.

Obviously a much wider protection must be given through the unimportunity of an Absorption Facile Place. We have only to call to mind the thick and layered
intestinal mucous membrane, with its epithelial lining as a buffer. Also the difficulty colonies have in settling on the healthy mucous membrane, and how few are found in the mucous plaques taken from the healthy gut. medically this fact is of great importance, pointing to the importance the local factor has in disease, and especially in disease of the intestinal tract, e.g., tuberculosis, where the germ is thought to often be swallowed and fail to produce the process without. But for example, given a fermentative animal, say in intestine of a child, then septicaemia, lues, and the colonies can settle. Also a ridution of continuity of epithelium or an inflammation will allow bacteria to settle in the wall or thence spread into the body. Pasteur found that when he fed animals with certain substances and substances, he could produce the disease very easily. His researches then enables one to lay still more stress on the "local predisposition" as an important factor in disease. But while the bacteria under ordinary conditions are not absorbed, their products can be of the researches detailed in the thesis help to make still more important the bacterial handling of disease and how a
Strict control, helped by a clear understanding of fermentative processes, should be exercised over Bacterii in the digestive tract, by means of drugs, etc. The Bacterii can develop poisonous out of animal constituents. Some diseases would seem to be intoxications from such fermentative products. This also proved by the increased separation of Fever in certain diseases e.g. Diphtheria. Now we can isolate the Bacterii, and examine their products and see nearer getting specific, against specific microbes - microbes as we know that the accumulation of certain meta-balies products of the Bacterii can directly destroy the originators.

The remedies detailed show that the or- ganic acids of the food have a not unimportant influence on Bacterii. They probably hinder development of fer- mentations, by killing or paralyzing the germs. It seems as if they could have the power to kill out extraneous bacteria, which have nothing to do with intestinal frauds, and to leave path free for usual fermentations. This may also explain their importance as depletors e.g. sour milk, vinegar, Pickles, mustard, cuminely too it might be said that where
abnormal fermentations, lactic or acetic acids might be given instead of hydrochloric acid. These acids also can probably excite the action of the bacteria, so that they are not capable of regeneration. And taking this in conjunction with the number of anaerobic bacteria found in the gut, one is brought again face to face with the local predisposition as a great factor in disease.

To conclude: These researches enable us to say that in the gastric juice and intestinal secretions we have very little protection against the organisms which find their way into the digestive tract— that the food acids are able to paralyze the germs, and hinder the development of fermentations, when these are not stopped by products of the bacteria themselves—and that in the intact walls of the intestine we have an efficient barrier against their absorption.

Allan Macfadyen, M. B. and
C. M. M. H. C. S. E.

University of Göttingen 10th May 1882
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3. Erhard, "Lehre von der Verdaunung." (Berlin, Arzneimittel 1879)


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9. Händel-Thimmesch. "Zur Pathologie der abdominalen Tetanie" (Centralblatt für klinische Medizin, 1885, No. 44, p. 737)

10. Weisser. "Beiträge zur Lehre des Lungen- und Rückenbeuge." (Freiburg i. Br. 1885)

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12. Sommerich. (Archiv für Hygiene, 1885)


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19. Malý: (Deutsches Physiologisches. Volume 5.)


22. Bulpini: "Ungiene chirurgica dei principii Biliari". (Rivista di Chimica medica. Turin 1884.)

23. Lindberg: "Die Gallens belagelhelf für Ersättnel. i muskulösen". (Uppsala Forhandlingsar 1884.)
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