## The Action of Salts and other Substances Used in the Curing of Bacon and Ham

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The art of curing bacon and ham is of great antiquity. As it is a traditional art, the methods employed have been passed on from generation to generation by practice and the spoken word rather than by the written word. As far back as 200 B.C., however, Cato the Censor wrote about the salting of hams. This recipe, like all old ones, only mentions salt, but nowadays saltpetre (KNO<sub>3</sub> or NaNO<sub>3</sub>) is also used, and sometimes other substances as well. Dry salting was the traditional method employed whereas nowadays bacon is usually pickle-cured. The underlying scientific principles of all forms of curing, however, remain unaltered, and it is necessary to know the nature of the raw material in order to understand the action of the salts used in curing.

This raw material consists of muscular tissue and fatty tissue held together by connective tissue, tendons and bone. Fatty tissue is relatively simple since it consists of connective tissue in which fat is laid down. In a carcass which is suitable for curing, the fatty tissue contains about 77-93% of chemical fat; at the most, therefore, the connective tissue represents only just over 20% of the fatty tissue, and may even be less than 10 % (Callow, 1938a). Curing agents, such as salt and saltpetre, are readily absorbed by the fatty tissue, although of course it is only the connective tissue and not the chemical fat which absorbs the salts. In fact, fatty tissue is easy to cure even by the most primitive of methods. The penetration of salt into bones, and into connective tissues and tendons is also a relatively simple matter. It is the muscular tissue which presents the curer with difficulties. Muscular tissue consists of very thin muscle fibres, which may be several inches long although their diameters are always microscopically small. These fibres are held together in bundles by connective tissue, and are interpenetrated with connective tissue which contains fat and behaves just like normal fatty tissue. Not only is the original micro-structure of muscular tissue complicated, but it is also affected by post-mortem changes. After death the reserves of glycogen in the muscle fibres change to lactic acid, and the pH of the muscular tissue is thus changed from well over 7.0 to 5.5 (Bate-Smith, 1939). The quantity of glycogen in the dead muscular tissue, however, depends on the type of muscle concerned and on the degree of muscular fatigue to which the animal was subjected shortly before death (Callow, 1939). Muscular fatigue depletes the glycogen reserves of muscles (Callow, 1938b) which, like the Psoas, move limbs, but it does not deplete to any marked extent the reserves of muscles which, like the Longissimus dorsi, have more of a fixational function (Callow, 1938b). A low reserve of glycogen (caused by fatigue) leads to a low production of lactic acid in the dead tissue, which therefore has a relatively high pH. In a case of extreme fatigue, the Psoas muscle had a pH of 6.7, and with less extreme cases of fatigue values over pH 6.0 are common (Callow, 1937).

The pH of muscular tissue affects curing in two ways. First there is the direct effect of the pH on the growth of bacteria, especially anaerobic bacteria. Ingram (1939) has shown that the growth of anaerobic micro-organisms responsible for taint in hams is

greatly diminished by a decrease of o I pH unit when once the pH drops below 6.0. Consequently these anaerobic bacteria fail to grow in muscular tissue when the pH is below 5.6. Secondly, the pH has an indirect effect. The microstructure of muscle, immediately after the death of the animal, is a 'close' one, i.e. the muscle fibres are full of fluid and there is little fluid between them. This state is characterized by a high electrical resistance both across and along the muscle fibres, the resistance across the fibres being considerably greater than it is along them. The production of lactic acid in the fibres, however, causes them to shrink and exude fluid and thus produce an 'open' structure. This state is characterized by a low electrical resistance, and the resistance across and along the fibres becomes nearly equal (Callow, 1937). Further, both salts and sugar penetrate into muscle with an open structure more rapidly than into that with a close structure (Callow, 1937). Moreover, the change from a close to an open structure cannot go to completion unless the pH is below 5.7 (Callow, 1936). In any case the change can be slowed down, though not prevented, by rapidly cooling the muscular tissue as soon as the animal has been killed (Callow, 1938c). An open structure with a low pH is advantageous, since the pH controls spoilage by inhibiting the growth of anaerobic bacteria, and the open structure hastens the penetration of salt, which in its turn helps to inhibit the growth of anaerobic bacteria, for Ingram (1939) has shown that about 5% of salt can prevent the growth of anaerobic bacteria at 37° even in the absence of acid.

The actual effect of salt and saltpetre on muscular tissue may be considered from two angles. First, the effect on the solubility of the muscle proteins, and secondly the behaviour of muscular tissue when immersed in solutions containing these salts. The solubility of the mixed proteins of muscular tissue increases in solutions of salt of up to about 9% (Callow, 1931) and then decreases. Potassium nitrate (Bengal saltpetre), when added to sodium chloride appears to have about the same effect on the solubility of proteins as an equimolar solution of sodium chloride (Callow, 1933). About half the protein (i.e. 10–11% of the weight of the muscular tissue) is soluble in solutions containing 6–9% of salt, but when the concentration of salt reaches 30% the solubility is only one-quarter of this value, i.e. about 2·5% of the weight of the muscular tissue (Callow, 1931, 1933).

The behaviour of muscular tissue in solutions of electrolytes and non-electrolytes is, however, more important than the solubility of the proteins. Muscular tissue, from the colloidal point of view, is a system of protein gel and protein sol. When a non-protein gel like agar is immersed in a salt solution, water is lost from the gel, and this loss increases as the strength of salt solution is increased (Callow, 1932). All systems of sols or gels containing protein, such as egg white in a collodion membrane, gelatin gels or muscular tissue, also lose water to salt solutions in the same way initially, but here the flow of water is later reversed, and ultimately there is a net gain in water. This is due to osmotic forces. The strong salt solution has a much higher osmotic pressure than the protein sol or gel, and water diffuses out as salt diffuses in. The protein-salt complex, which is formed, however, has an osmotic pressure greater than that of the salt solution, and the outward flow of water is therefore reversed and water flows inwards. Proteins cannot form such complexes with sugar, and for this reason

a system containing protein loses water to a sugar solution and, moreover, the water never flows back again as is the case with a non-protein gel like agar immersed in a salt solution (Callow, 1932).

But muscular tissue is not just a protein gel, since it has a complicated microstructure, and there comes a point at which this structure affects the behaviour of muscular tissue immersed in salt solutions. Thus, if a gelatin gel is immersed in solutions of salt of increasing strength, it swells to a definite extent, the uptake of

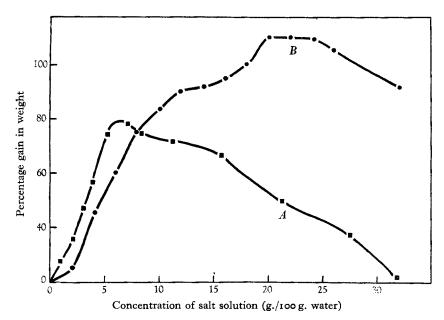


Fig. 1. Swelling of pork muscle at 0° on: A immersion in solutions containing various amounts of salt, and B immersion in a 2% solution of salt which was progressively increased in strength (Callow, 1933). Crown copyright.

water depending on the salt content of the solution. Even if the gel is placed in a series of salt solutions of increasing strength, the uptake is eventually the same. With muscular tissue, however, the actual amount of swelling depends upon the manner in which the immersion is carried out (see Fig. 1). By gradually increasing the strength of salt solution it is possible to obtain a much greater gain in weight in strong salt solutions (over 10% of salt) than is possible when the muscular tissue is immersed in the final salt solution straight away (Callow, 1933).

The swelling of such systems as muscular tissue and gelatin gels in salt solutions is controlled by two opposing forces: (a) a swelling pressure developed by the osmotic forces of the protein-salt complexes, and (b) a counter pressure due to the elastic forces of the gel structure. Fig. 1 shows that the heterogeneous structure of muscular tissue has less resistance to swelling when the initial loss of water is minimized by starting with dilute solutions and gradually increasing their strength than when the initial loss of water is greater because the tissue is immersed straight away in strong solutions of salt. This is presumably because the fibres are not held firmly together and can slip

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past one another or be pushed apart from one another. Muscular tissue thus behaves in some ways like a simple protein gel, but extra effects due to the complex microstructure must also be taken into account when considering the effects of the salts and other substances used in curing.

In curing, the diffusion of the curing agent into the muscular tissue and also the diffusion of water and other substances out of it must be considered. During curing the initial outward flow of water, proteins and other material is soon reversed; but the slower the diffusion inwards, the longer the outward flow can occur. Slow inward diffusion is helped by immersion of the muscular tissue in relatively weak concen-

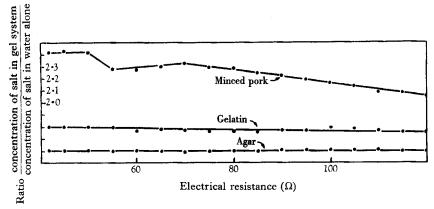


Fig. 2. Effect of increasing the concentration of salt (i.e. decreasing the electrical resistance) on the ratio of the concentration of salt in the gel system to that in water alone, at the same electrical resistance. Salt solution expressed as g. salt/100 g. water (Banfield & Callow, 1934).

trations of salt, by a close microstructure of the tissue, and, oddly enough, by the use of solid salt instead of a solution. With solid salt and with the strong pickles used in curing, the surface of the muscular tissue is rapidly changed, owing to the precipitation of some of the soluble proteins; this minimizes the loss of protein but not the loss of water from the muscular tissue. For this reason there is surprisingly little protein  $(o\cdot 4\%)$  in pickle in which many batches of bacon sides have been immersed (Callow, 1934).

When once salt has diffused into muscular tissue, or even when minced tissue and salt are mixed together, the microstructure is altered. The texture becomes much more jelly-like, and the muscle juice is more firmly held. Thus, when 100 g. of lean pork were mixed with ether, it was possible to express by mechanical pressure 60.7 g. of water out of a total of 72.8 g.; whereas with 100 g. of lean bacon only 25.5 g. out of the 63.1 g. of the water could be so expressed (Callow, 1927a). Thus with uncured muscular tissue over 80% of the water could be expressed but with cured tissue only 40%. What is the explanation of this? It appears probable that salt makes each individual muscle fibre swell up and hold water like a miniature gel system. The evidence for this is twofold. First, the electrical resistance of minced muscular tissue to which known amounts of salt have been added varies with the salt content, in a manner which is different from the variation shown by agar and gelatin gels containing the same proportion of salt (Banfield & Callow, 1934, 1935). At all electrical

resistances the salt content of the gels is greater by a constant factor than the concentration of a solution of salt of the same electrical resistance, whereas, with muscular tissue, this factor increases as the salt content is increased, i.e. as the electrical resistance is diminished (see Fig. 2). This might be expected to happen if the 'open' structure of the muscular tissue became progressively more and more 'closed' as the salt content was increased. Secondly, it can be shown that the salt flavour of salted muscular tissue and of bacon itself, is not directly related to its actual salt content (Ingram, 1947) and may alter with time in the same piece of bacon, becoming saltier or even less salt during maturation and storage. If we make the highly probable assumption that only part of the salt present in bacon has time to affect the palate before the bacon is swallowed, and that the salt within the fibres is less likely to diffuse to the palate than the salt between the fibres, we see why there can be variations in the flavour of bacon which are not parallel with the total salt content.

One factor in the curing of bacon and ham is the growth of micro-organisms on the surface of the meat. Only a limited range of micro-organisms can grow because of the selective action of salt (see Fig. 3). This, as Ingram (1934) has found, depends upon the concentration of the salt, which in its turn depends on how much water has been removed by curing or afterwards by drying and smoking, as well as on the quantity of salt which is present. The extreme case is where the surface is so dry that nothing grows. On a moderately dry surface, as with the York ham, moulds grow, and the growth of a green mould-probably a Penicillium—is the final stage in curing and maturing a York ham. The growth of mould inhibits the growth of bacteria and also diminishes the risk

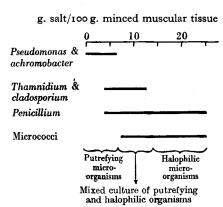


Fig. 3. The selective action of sodium chloride on the growth at o° of various classes of micro-organisms on the surface of minced muscular tissue (Ingram, reported by Callow & Moran, 1938).

of the fat going rancid; instead, the fat is hydrolysed (Lea, 1933) into fatty acids and glycerol. With Wiltshire bacon, which is wetter than York ham, the surface becomes overgrown with a film of micro-organisms in the maturing cellar. The tank pickle also contains micro-organisms. In both cases the saltpetre used in curing is reduced to nitrite, which then reacts with the reduced haemoglobin of the muscular tissue to give nitrosohaemoglobin, the pigment to which both bacon and ham owe their colour (Brooks, 1937). It has been suggested that the presence of nitrite is responsible for the cured flavour of bacon, and that bacon could be produced in the absence of micro-organisms, provided nitrite was present as well as salt (Brooks, Haines, Moran & Pace, 1940). This is doubtful, however, because pickled pork contains nitrite in sufficient quantities to give a cured colour and yet it lacks that subtle extra flavour which one associates with fully matured bacon.

The special flavour of fully matured ham depends upon hydrolytic and oxidative changes in the fatty tissues, in addition to the changes in the muscular tissue which

give rise to the characteristic flavour of both bacon and ham. Extra flavours in both bacon and ham can be obtained by using sugar and spices, beer or vinegar as curing agents. Sugar acts by removing more water than salt and is changed by the action of micro-organisms to acid. The most important extra agent, however, is wood smoke. Smoking both dries the bacon or ham and impregnates it with a whole range of substances which act as antiseptics and give it an extra flavour. It is not always realized that among the substances in wood smoke is formaldehyde (Callow, 1927b), and that the smoke also contains a wide variety of other substances, e.g. acetaldehyde, furfuraldehyde, 5-methyl furfuraldehyde, diacetyl, acetone, methyl and ethyl alcohol, acetic and formic acids, and phenols, as well as tar and wax (Pettet & Lane, 1940). No wonder that wood smoke imparts an added flavour to bacon and ham. There is, however, another function of smoking. It causes the bacon or ham to lose from 3 to 10% of its weight by evaporation of water; smoking thus involves drying. Moreover, some of the substances in wood smoke are anti-oxidants, and the fatty tissue of smoked bacon, therefore, goes rancid less readily than that of unsmoked bacon (Lea, 1933).

## REFERENCES

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Banfield, F. H. & Callow, E. H. (1934). Rep. Food Invest. Bd, Lond., p. 72. Banfield, F. H. & Callow, E. H. (1935). J. Soc. chem. Ind., Lond., 54, 413 T.
Bate-Smith, E. C. (1939). J. Physiol. 96, 176. Brooks, J. (1937). Proc. roy. Soc. B, 123, 368.
Brooks, J., Haines, R. B., Moran, T. & Pace, J. (1940). Spec. Rep. Food Invest. Bd, Lond., no. 49.
Callow, E. H. (1927a). Rep. Food Invest. Bd, Lond., p. 17.
Callow, E. H. (1927b). Analyst, 52, 391.
Callow, E. H. (1931). Rep. Food Invest. Bd, Lond., p. 144.
Callow, E. H. (1932). Rep. Food Invest. Bd, Lond., p. 101.
Callow, E. H. (1933). Rep. Food Invest. Bd, Lond., p. 97.
Callow, E. H. (1934). Rep. Food Invest. Bd, Lond., p. 65. Callow, E. H. (1936). Rep. Food Invest. Bd, Lond., p. 75.
Callow, E. H. (1937). Rep. Food Invest. Bd, Lond., p. 46.
Callow, E. H. (1938a). Rep. Food Invest. Bd, Lond., p. 45. Callow, E. H. (1938b). Rep. Food Invest. Bd, Lond., p. 53. Callow, E. H. (1938c). Rep. Food Invest. Bd, Lond., p. 55.
Callow, E. H. (1939). In the Press.
Callow, E. H. & Moran, T. (1938). J. R. sanit. Inst. 58, 157.
Ingram, M. (1934). Rep. Food Invest. Bd, Lond., p. 77.
Ingram, M. (1939). In the Press.
Ingram, M. (1947). Personal communication.
Lea, C. H. (1933). J. Soc. chem. Ind., Lond., 52, 57 T.
Pettet, A. E. J. & Lane, F. G. (1940). J. Soc. chem. Ind., Lond., 59, 114.
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