Porcine Meat Inspection

Anatomy, physiology and disease conditions

A Grist

CONTEXT

CONTENTS

INTRODUCTION

This book forms part of a series that has been designed to provide a study aid for students undertaking the Royal Society for the Promotion of Health Red Meat Inspection Certificate and other courses where knowledge of porcine meat inspection is required such as those for Environmental Health Officers and Official Veterinary Surgeons. It is hoped that it may also provide information in a 'user friendly' format for abattoir owners and producers as to the nature of rejections recorded by inspectors.

Wherever possible, photographs illustrating anatomical features and conditions have been included, the latter generally depicting severe examples of conditions. This is a conscious decision, arrived at after giving numerous lectures on meat inspection where I learnt that no two people would describe something the same way.

The section covering anatomy and physiology does not go into great detail; numerous other texts fulfil this function. The order that the bodily systems are introduced should not be construed as an order of importance. The section covering diseases loosely follows the format of name, synonym, aetiology, pathogenesis, clinical lesions, gross lesions and judgement as to the fitness for human consumption. The diseases are listed in alphabetical order for ease of reference.

The Affections of Specific Parts and Conditions Encountered at Post-Mortem Inspection in Abattoirs is hoped to provide a useful tool for day-to-day inspection decisions in the abattoir environment.

AUTHOR DISCLAIMER

I must add that the judgements as to the fitness for human consumption given in this book are my own views, gained through experience and consultation with others.

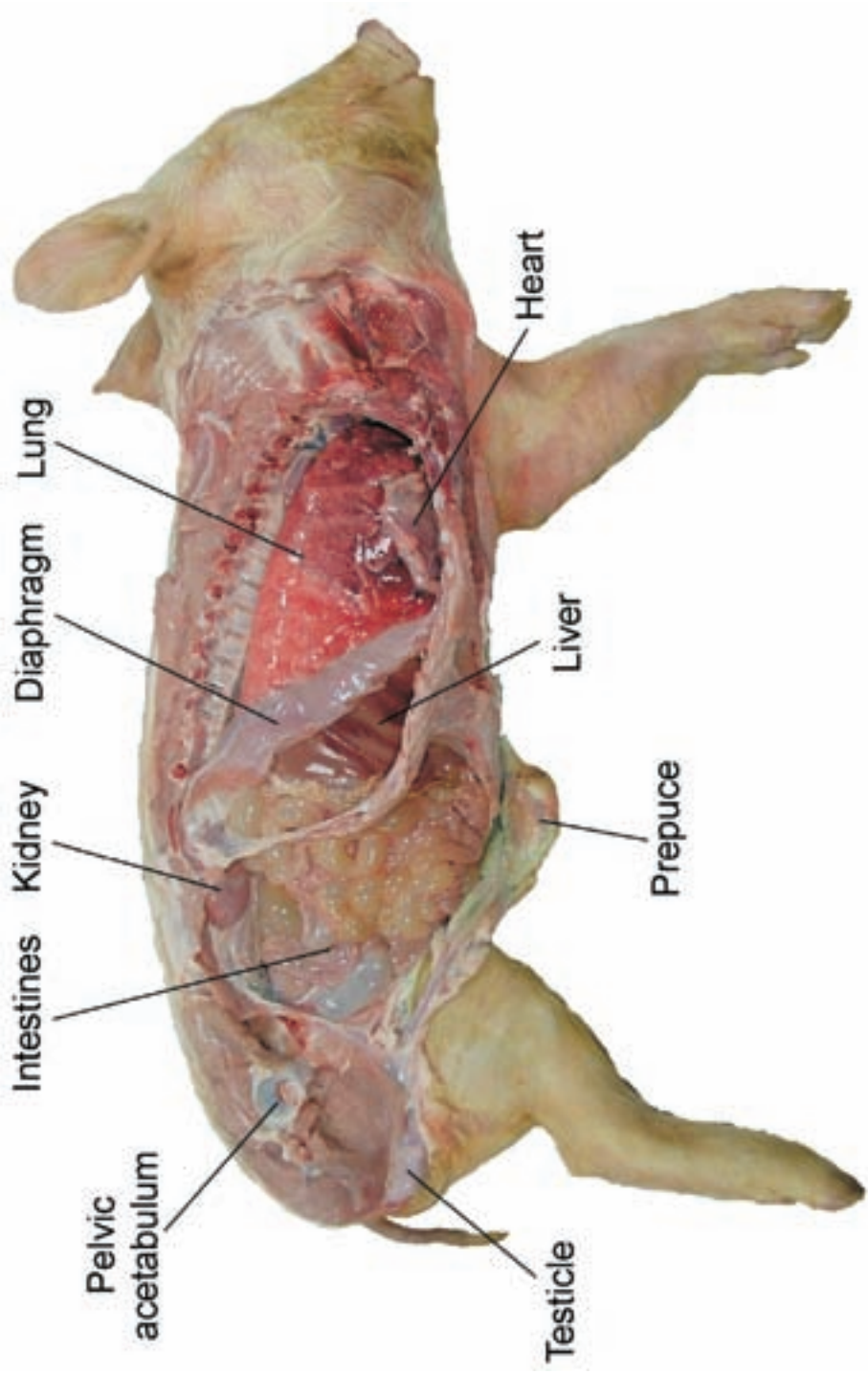
A.Grist MAMI

DEDICATION

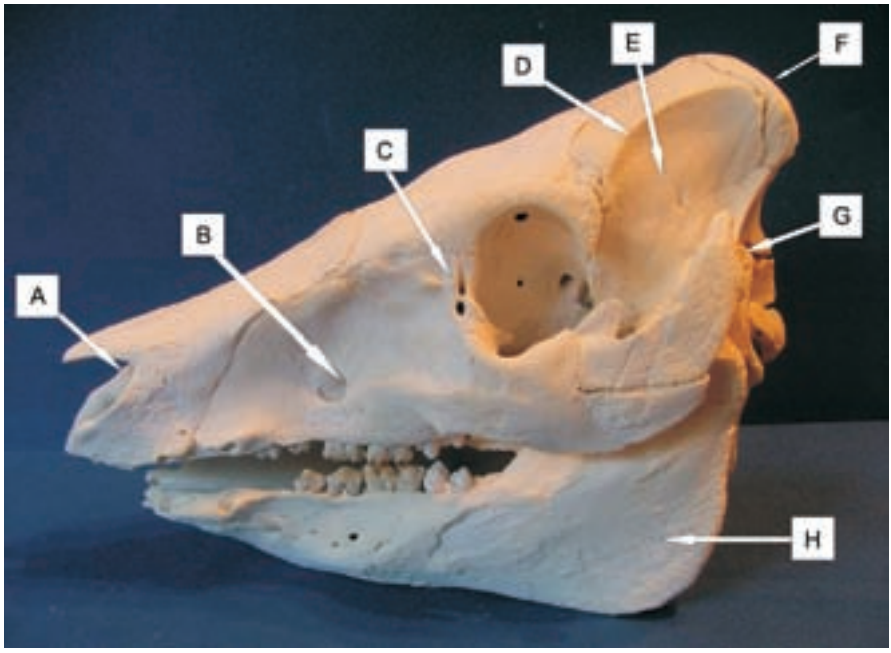
This is the fourth book I have produced, but the dedication remains the same as in previous volumes. I have always held the opinion, during my time as a Meat Hygiene Inspector, abattoir manager and lecturer that the food that I inspect should be fit for my own children to eat. If you would not feed it to your own children, you should not expect the consumer to feed it to theirs.

To that end I dedicate this to my wife Grace and my children George, Elizabeth, Henry and Harriette.

In memory of my Grandfather, Ronald Maryan. Forever in our thoughts.



Partial dissection of porcine carcase – right side view

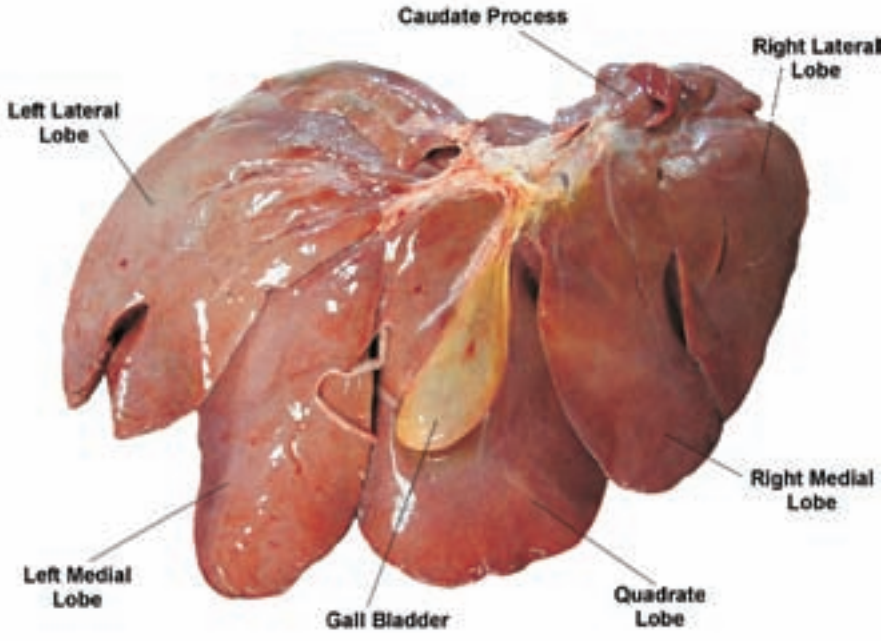


The Porcine skull. A-Nasoincisive notch, B-Infraorbital foramen, C-Lacrimal foramina, D-Temporal line, E- Temporal fossa, F-Nuchal crest, G- External acoustic meatus, H-Mandible

THE ROSTRAL BONE

An adaptation in swine that is not present in other meat animals is the rostral bone. This is situated caudal to the nasal septum and in addition to support of the snout, provides an anchorage for the nasal cartilages and the main muscle that moves the snout.

The rostral bone in situ



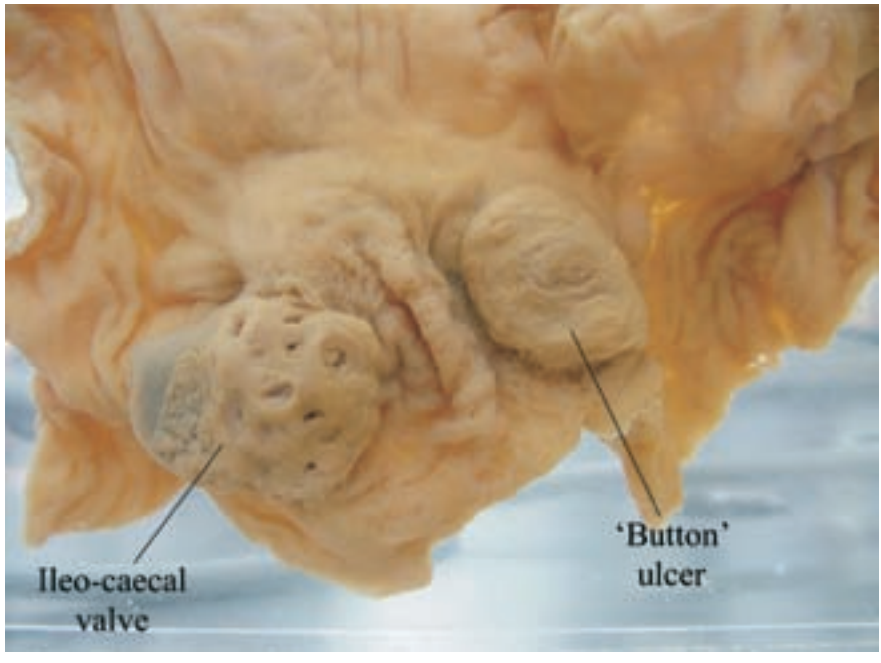
Normally covered by the pancreas is situated the entry point of the portal vein and artery into the liver, surrounded by the hepatic lymph nodes, and also indicates the point at which the cystic duct leaves the liver for the gall bladder.

LIVER STRUCTURE

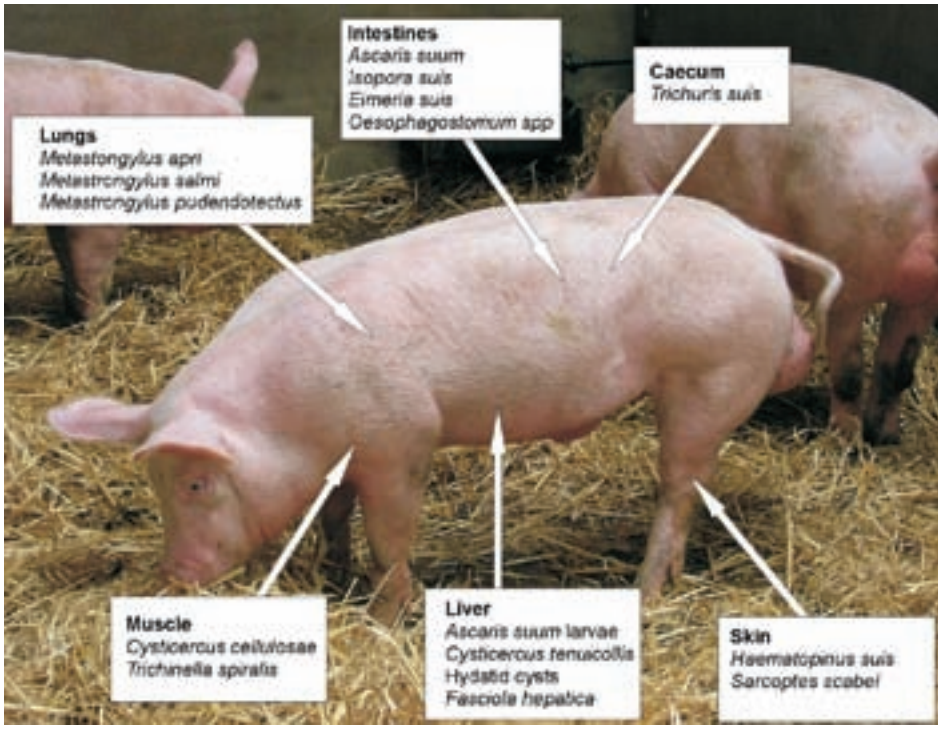
The liver is mainly composed of small filtration and secretion units called lobules. Each lobule is enclosed in thin connective tissue which in the pig is fibrous and comprises specialist liver cells, including the main liver cells, hepatocytes, surrounding a central capillary of the hepatic portal vein. The hepatocytes are segregated from the blood vessels by a basement membrane covered with basic epithelial cells and the phagocytic Kupfer cells. The hepatocytes are closely packed together but do have small canals between them that collect the produced bile and then connect together to form a network that eventually produces bile ducts. The primary cystic duct exits the liver at the portal notch and transports the bile to the gall bladder where it is stored and concentrated.

SWINE FEVER

Synonyms:	Hog cholera, Classical swine fever
Type	Viral
Aetiology	Togavirus
Pathogenesis	Infected animals shed virus in all body secretions especially urine. Infection route upper respiratory or upper digestive tract. Initial multiplication occurs in local lymph nodes and tonsular material, followed by viraemia and localization in white blood cells of lymphatic system and blood circulation, the spleen, visceral lymphoid tissue and bone marrow. Proliferation of endolethial cells follows leading to occlusion of the vessels.
Clinical	Fever, reddened areas of skin. 'Dog sitting' position. Huddling. Goose stepping gait when moved. Vomiting and constipation.
Gross	Enlarged 'strawberry' lymph nodes due to haemorrhage. Necrosis of tonsular material. Renal petechiae, ecchymosis and petechial haemorrhages in skin, lung and bladder. Splenic infarction in margins of the spleen. Button ulceration of proximal colon following blood vessel damage and subsequent necrosis of the area and the development of concentric rings of necrotic material.
Judgement	Unfit for human consumption **Notifiable in UK**



Button ulcer in colon (Sample fixed in formalin)



Main parasites of pigs

PROTOZOA

Protozoa are single-celled organisms that, unlike bacteria, possess a nucleus and other organelles that enable them to lead an independent existence. Protozoa are mobile organisms, using methods of propulsion such as flagella, cilia and undulating membranes. They feed by enveloping particles and digesting them, followed by the extrusion of waste material from the cell.

COCCIDIOSIS

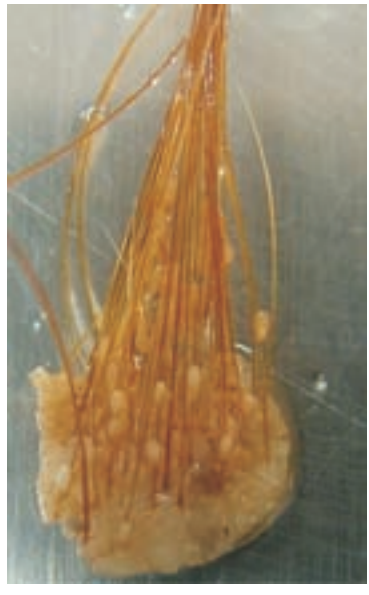
Coccidiosis is a disease condition caused by the actions of the coccidian protozoan parasites *Isopora suis* and *Eimeria suis* of which the former tends to be the most prevalent This is an intercellular parasite of the cells that line the internal surface of the intestines (epithelial cells) causing lesions by destroying the cells as part of their lifecycle. The severity of the disease is dependant on the number of infective agents ingested, but an infected individual can pass several hundred million of these agents in faeces during the course of the disease.

ECTOPARASITES

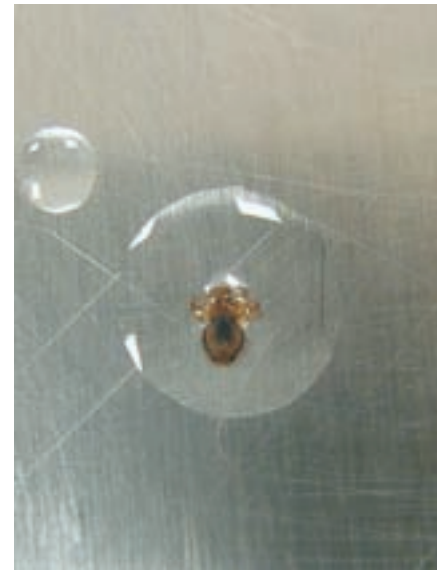
LICE

Haematopinus suis is the main louse encountered on pigs; it is host specific and is classed as a 'sucking louse'. Adults are approximately 0.5cm in length, possess strong claws for anchoring to bristles and have a predilection for thinner areas of skin such as the throat, skin folds and inner thighs where they feed on the blood of the pig. Feeding is undertaken up to six times a day at a new bite site where the specialised mouthparts burrow into the skin and anchor the louse and the stylets (mouthparts) burrow through tissue seeking a blood vessel, usually a venuole.

H.suis has a lifespan of approximately 35 days as a permanent parasite of the host, if it leaves the host it has a survival time of 2-3 days in the environment. Females lay eggs (nits) attached to the bristles (3 to 6 eggs per day for 25 days). These hatch and the nymphs usually migrate to the ear canal. The nymphs undergo 3 moults in which they develop into the adult form usually within 10 days at which point both male and females reach sexual and reproductive maturity.



'Nits' – lice eggs attached to the bristles of a Tamworth pig



***Haematopinus suis* - Mature louse in formalin droplet**

The affects on the host of *H.suis* infection can be severe in young animals with anaemia being the most obvious clinical manifestation. In older pigs,

PORCINE STRESS SYNDROME

Also known as Enzootiche herztod, Porcine Malignant Hyperthermia, Transport myopathy, Pale Soft Exudative Pork and PSE. Porcine Stress Syndrome affects well muscled breeds such as the Landrace, Duroc, Large White, Hampshire and Yorkshire. The presence of the recessive Halothane gene predisposes these breeds, especially the Landrace, to stress and stress-related death, normally associated with environmental stressors such as transportation to the abattoir.

Clinically the onset of PSS is characterised by rapid breathing, muscle and tail tremor, skin blotches and elevated temperature, followed rapidly by death. The recessive gene produces biochemical abnormalities within the muscle altering the metabolism from an aerobic to anaerobic state, increasing tissue acidity. Once slaughtered, animals affected by PSS develop rapid rigor (5-10minutes as opposed to hours) and the pH of the muscle falls rapidly to below 6.0 usually within 45 minutes, the ultimate pH being the same as normal.

PSS produces Pale, Soft, Exudative Pork, which although technically fit, has poor keeping qualities. PSE pork is due to the rapid pH fall whilst the carcass is still hot leading to a breakdown of the myosin heads within the muscle which compresses the actin/myosin element. This has a threefold effect on the meat from these animals. Firstly there is greater light deflection due to the increased myofibril density which manifests as a paleness of the meat. Secondly the compression forces water from the myofibrils which accumulates and disrupts the integrity of the connective tissues which softens the meat. Thirdly this accumulated water between the myofibrils drains during butchery of the meat leading to exudation, a drip loss from the meat can be 5% higher than normal.

A piece of PSE pork compared to normal

Lungs

- 2-3 left lobes
- 3-4 right lobes
- Marked surface lobulation
- Accessory bronchus to right lobe
- Weight: 300-450g

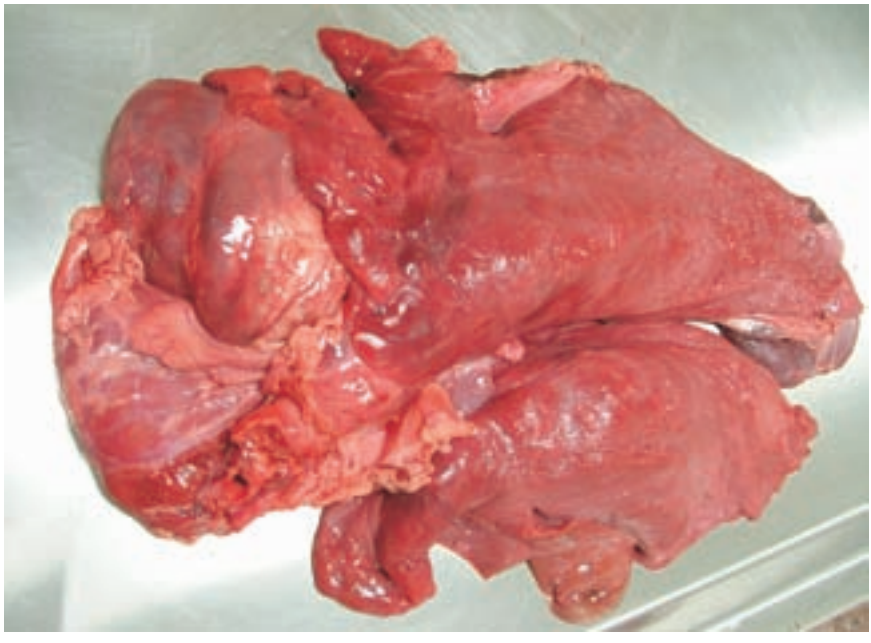
Heart

- Fat is soft and greasy due to presence of olein
- 2 ventricular furrows
- 4 chambers; left atrium and ventricle, right atrium and ventricle
- Weight 0.25-0.4Kg

Spleen

- Tongue/strap shaped
- Omentum attached to longitudinal ridge
- Triangular in cross section
- White pulp visible in cut surface
- Weight 0.2-0.25Kg

LESION	POSSIBLE DIAGNOSIS
LUNGS (contd.)	
Congested, oedematous	Haemophilus pleuropneumonia
Consolidation of anterior lobes	Enzootic pneumonia, Necrotic enteritis
Dark red	Blood congestion
Dark red raised patches in diaphragmatic lobe	Haemophilus pleuropneumonia
Emphysema	Swine influenza
Exudative pneumonia	Pasteurellosis
Fibrinous pleurisy	Mycoplasma, Glässer's disease, Haemophilus pleuropneumonia, Swine influenza
Oedema of interlobular septa	Haemophilus pleuropneumonia
Petechial haemorrhages	Swine fever
Pneumonic and hepatised	Salmonellosis - acute enteric, PRRS, Swine influenza
Pulmonary oedema	Aujeszkys disease, Necrotic hepatitis, Swine influenza
Small, dark red areas in anterior lobes	Enzootic pneumonia, actinobacillosis, Pasteurellosis
Solid and haemorrhagic	Haemophilus pleuropneumonia
Yellow fibrinous pleurisy	Haemophilus pleuropneumonia
Yellow, pus-filled nodules	Actinomycosis, PRRS



Hepatized pneumonia

Index

C

D

E

F

G

H

I

J

K

L

M

N

O

P

R

S

T

U

V

W

Z

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Produced primarily for students undertaking qualifications in red meat inspection, this fully illustrated book guides the reader through pig anatomy and physiology before explaining the cause and nature of disease. Further chapters graphically describe diseases and conditions encountered in pigs including parasites and tumours, and include a chapter on conditions found at the abattoir with suggested judgements as to fitness for human consumption. Revision guides covering anatomy, condition and cause and a basic diagnostic aid complete the volume.

The full colour photographs make this an invaluable tool for all those for whom knowledge of porcine anatomy, diseases and other conditions is required, including veterinary surgeons and meat inspectors within the abattoir, and also pig producers who will be receiving condemnation data from these establishments.

Contents

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CONTEXT