LABOKLIN
LABORATORY FOR CLINICAL DIAGNOSTICS

Directory of Tests
Horse

2025/26

www.laboklin.com

As of December 2024

Foreword

Dear colleagues

the good is the enemy of the better – we have updated our horse directory of tests and are sending you this new version today. Numerous additional tests and profiles have found their way into the booklet and thus also into our submission forms. We hope you enjoy reading it.

Bacteriology: In addition to species-specific tests and antibiograms or symptom-orientated PCR profiles, we also offer hygiene monitoring for the veterinary practice. Additionally we produce and distribute autovaccines, an alternative therapeutic concept when antibiotics are not or no longer effective.

Histology and Cytology: Whether BAL, cytology or histological assessment of biopsies, we work on a species-specific basis and have a team of more than 20 pathologists.

Genetics: Genetics is not just a breeding basis for us – genetics is also the clarification of differential diagnoses, such as PSSM. Enjoy the unique combination of genetics and veterinary laboratory.

Clinical Chemistry: Certainly, we offer general profiles, but we are also strong in endocrinology. We set up tests methodically in a way that in many cases minimises the pre-analytics impact.

No matter if oestradiol or testosterone, AMH or luteo-placental shift, you get reliable and reproducible results that you can count on in your further work.

The Plus with Laboklin:

- You can reach us 24/7 via MyLab. Further enquiries about findings, courier registrations, reorders – everything is in good hands with us.
- The 4Paws APP takes over the burden of reminding owners about regular tasks, such as deworming or vaccinations.

Our team of horse specialists will be happy to answer any questions you may have.

Further education is not overlooked either: You will find numerous events at the Laboklin Academy website.

We look forward to hearing from you!

Best regards

Dr Elisabeth Müller
CEO LABOKLIN GmbH & Co. KG

Abbreviations and Notes

Test methods

AAS	atom absorption spectrometry
CEDIA	cloned enzyme donor immuno-assay
CFT	complement fixation test
CLIA	chemiluminescence assay
EIA	corresponds to ELISA
ELISA	enzyme linked immunosorbent assay
cELISA	competitive ELISA
GCMS	gas chromatography – mass spectrometry
HAH	haemagglutination inhibition test
HPLC	high performance liquid chromatography
ICPMS	inductively coupled plasma mass spectrometry
IFAT	indirect fluorescent antibody test
LCMS	liquid chromatography – mass spectrometry
MAT	microscopic agglutination test
MS	mass spectrometry
PCR	polymerase chain reaction
RIA	radioimmunoassay
SAFC	sodium acetate-acetic acid-formalin concentration
SAT	slow agglutination test
VNT	virus neutralisation test

Sample material

BAL	bronchoalveolar lavage
CP	citrate plasma
CSF	cerebrospinal fluid
EB	EDTA blood
EP	EDTA plasma
HB	heparin blood
HP	heparin plasma
NaFB	sodium fluoride blood
S	serum
TBS	tracheobronchial secretion

* partner laboratory

Duration of examination

- applies from receipt of the sample in the laboratory in Bad Kissingen
- working day: usually Monday – Friday; for findings that are also transmitted on Saturday, this is indicated.

In our "Directory of Tests – Horse" we have compiled specific equine tests and procedures. Basic information concerning pre-analytics, haematology and clinical chemistry, which are fairly similar for all species, can be found in our detailed Compendium.

Tests offered may be subject to change.

Contents

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1. Sampling and Shipping – Pre-analytics

The first step in the process of examining a sample is the pre-analysis. Pre-analysis includes all steps from patient preparation, specimen collection and transport of the sample to the lab to the preparation of the sample for analysis.

Preparation of the patient
Fasted blood samples are not unproblematic in horses and only indicated for special tests, e.g. insulin and glucose for diagnosis of Equine Metabolic Syndrome (EMS).
The owner should be informed about the influence of physical activity or stress on blood test results: particularly muscular enzymes but also glucose and lactate can show elevated serum levels.

Labelling
The names of the animal and the owner should be clearly marked on the submission form and the sample.
Alternatively, LABOKLIN bar code labels can be used. For samples that will be tested bacteriologically, cytologically, or histopathologically, it is important to indicate the sampling site. For function tests, it is necessary to specify the time of collection or number the samples consecutively.

Sample material?
Details on the recommended material can be taken from our test descriptions or the submission forms.

Microbiology
It is important to collect samples as sterile as possible to avoid contamination.

Bacteriology and mycology: swab with transport medium

PCR: swab without transport medium (exception: Taylorella equigenitalis/CEM Profiles – swabs with Amies medium are required), whole blood (EDTA), fresh organ specimen (depending on pathogen)

Urine: sterile tube, swab with medium or Uricult

Hair/skin scrapings: sterile tube

Faeces: standard faeces tube

Puncture specimen: sterile tube, swab with medium (for bacteriological examination)

Blood: blood culture flask (written order required, subject to a charge)

Histology/cytology
When submitting tissue samples for histopathological examinations, the following points should be observed:
- Biopsies should have a minimum size of approx 5 mm (excluding transendoscopic biopsies), care must be taken not to spoil small biospies by crush artefact.
- Immediate fixation (4 % formaldehyde ≙ 10 % formalin)
- Submission of an anamnesis including clinical findings and objective

Surgical biopsies should be representative of the clinicial lesions and ideally be free of preparation artefact (e.g disruption, squashing, electrocoagulation). For multiple or disseminated lesion, multiple samples may be obtained (for accounting, see price list/ submission form).

- Skin punches
Skin punch biospies should have a minimum diameter of 6 mm. The biopsy site should not be prepared surgically (e.g. shaving/scrubbing) as this may remove diagnostic lesions. For best results it is advised to sample multiple primary lesons (minimum of 3 skin punches).

To aid meaningful histological examination the sample should be accompanied by a concise clinical history including clinical findings and differential diagnosis.

- Cytology
Samples can primarily be taken as impression smear, scraping or aspirate (e.g. synovial fluid) with or without suction. Fine-needle aspiration is done using a 22 – 27 gauge needle attached to a syringe. A vacuum may be created and, if possible, the tissue should be punctured several times in different directions. To avoid the aspirate receding into the syringe, the vacuum must be released before detaching the needle. The material obtained is subsequently expressed onto the side of a glass slide. A second slide is placed flat at a right angle on top of the first one and is then carefully pulled away across the slide to spread the material. If the sample is more liquid, a steeper angle – like in a blood smear – should be applied.

For the cytological examination of aspirates, excretions or secretions, the fluids obtained are sent in EDTA test tubes. Ideally, the fluid obtained can already be centrifuged in the practice (2500 – 3000 rpm for 3 to 5 minutes).
Additionally, the supernatant is decanted and the sediment is carefully spread like a blood smear and shipped air-dried. If the aspirates are sent directly, EDTA tubes should be used.
For bronchial, conjunctival and vaginal cytology, the swab obtained (cytobrush) should be rolled onto a glass slide, not smeared.
All smears should generally be sent in air-dried, but unfixed and unstained. The most important point is to create a thin smear consisting of only single layer of cells (monolayer). Cytological preparations which are too thick are of limited diagnostic value.

Molecular Genetic Analysis of hereditary diseases, coat colour, performance and identity
An EDTA blood sample (approx. 0.5 ml) is required to perform these genetic tests. For horses, it is also possible to send in hair samples with roots (approx. 20 plucked mane or tail hairs), but only a limited amount of DNA can be extracted from them. For all partner laboratory services (except speed gene) only hair samples are to be submitted.

EB (purple top)
For doing a blood count, EDTA blood is the most suitable material. After collecting the sample, tilt the test tube carefully several times to mix the blood with the anticoagulant.

For the determination of clinical chemical and/or serological parameters it is only second choice, since EDTA as an "ion capturer" interferes with a variety of analyses.

- Haematology
We recommend to send an air-dried blood smear for the differential blood count along with the EB sample. This way, blood cells are fixed and alterations caused by ageing of the blood are avoided. If the smear is done using EDTA blood: please tilt the tube carefully a few times first before taking a drop from the sample to make sure that the composition of cells in the smear represents the composition of the whole blood.

- PCRs from whole blood
EDTA is the most suitable anticoagulant; heparin in contrast may inhibit the PCR.

Serum (red top)
With only a few exceptions, serum is the universal sample material for the determination of clinical chemical, serological and endocrinological parameters. Before separating the serum from the clotted blood, coagulation should be completed (allow to stand for at least 30 – 60 minutes before centrifugation; see above for separation process). If non-centrifuged samples are sent, haemolysis caused by transport and ageing might limit the diagnostic value of some parameters. Especially LDH, CK and K are often elevated because of the delay in sample processing in mobile practices. This needs to be taken into account when interpreting the test results.

Plasma
Plasma samples are drawn into tubes with anticoagulants (EDTA = purple top; heparin = green; citrate = blue; fluoride = grey). Please note: the additives may limit the number of analyses; with the exception of PCRs, heparin samples can be used for virtually all purposes.
The samples can be centrifuged immediately after collection (see above).
In citrate samples for the determination of coagulation parameters, the mix ratio of 1:10 is mandatory (please observe the marking line at the edge of the tube).

For exact glucose and lactate values, the additional submission of sodium fluoride blood is necessary. Both parameters are only stabilised reliably by fluoride (centrifugation as early as possible to separate the whole blood from the plasma).

Factors interfering with analysis

- Haemolysis
Haemolysis is caused by leakage of intracellular components of the erythrocytes such as iron, potassium and especially haemoglobin due to damage of the cell membrane. Haemoglobin causes a red colouration of serum/plasma which primarily interferes with the photometric testing done in clinical chemistry.
In horses, the parameters that are most susceptible to haemolysis are potassium (K), LDH and CK.

	Parameter	
haemolysis	LDH, CK, AST, bilirubin, creatinine, PO4, K, Fe, fructosamines	⬆
haemolysis	Ca, Mg, glucose	⬇

- Lipaemia
Lipaemia in horse blood samples is very rare: hyperlipaemic syndrome especially in ponies or with pronounced Cushing's disease.
Lipaemia refers to the milky/turbid discolouration of serum/plasma caused by triglycerides. Lipaemic samples may complicate the measurement of several clinical parameters.

- Icterus
Icterus is the yellowish discolouration of serum/plasma due to excess amounts of bilirubin in the blood. The most common icterus in horses is starvation icterus which already develops after short periods of fasting/anorexia. It must not be mistaken for the physiological yellow colouration of serum/plasma in horses due to physiologically high bilirubin concentrations! If hyperbilirubinaemia is excessive, the determination of some parameters may be complicated.

- Medication
Numerous drugs may influence clinical chemical or haematological parameters; listing them all would exceed the volume of this manual. When interpreting laboratory results, possible effects of the drugs in pre-treated horses should be taken into account.

Packaging and transport of samples

Please remember to pack your shipment according to EU regulations (European Agreement concerning the International Carriage of Dangerous Goods by Road, ADR, and International Air Transport Association, IATA):
Generally, sample tubes should be placed in break-proof transport containers that contain absorbent material for leakage protection and then packed, together with the submission form and cushioning material (not provided by the lab), in the transport (courier) box (min. dimension of 100x100x100 mm). Volume restriction: sample of (total) 1000 ml (applies to liquid samples) or a total weight of 4 kg (applies to illiquid samples). LABOKLIN provides such protective outer packaging free of charge.

There are 2 possible categories of samples. The outer package needs to be tagged according to the respective category with the labels shown in the illustration below.
An exempt animal specimen is a patient sample for which there is minimal likelihood that pathogens are present (e.g. formalin-fixed tissue samples).
Classification must depend on professional judgement which is based on the anamnesis, the signs, the patient's individual circumstances and local endemic conditions. In case of doubt, it is recommended to ship as infectious substance of Category B.
Infectious substance category B samples (e.g. swabs for germ detection, faeces, etc.) must be marked as "Biological substance, Category B" and "UN 3373"; while the specification "UN 3373" needs to be in a rhomb of at least 5 cm x 5 cm of size. The edge of the rhomb must be at least 2 mm wide and the letter height of both specifications must be at least 6 mm. The marking has to be weather-proof.

Important:
The sender is liable for the goods to be transported (i.e. sender is liable to recourse in case of damage/costs caused by samples that are not properly packed).
If requirements are not met, there is a risk of your shipment being returned to you by the courier company.

Please do not to leave any needles in the sample tubes!
Do not seal the tubes!
If protective covers/transport containers are used, the lids will remain securely closed.

For shipment from a non-EU country, please contact LABOKLIN in advance.
Should you have any other questions, please do not hesitate to contact your local LABOKLIN representative or contact us directly: service@laboklin.com

Sample preparation/Shipping supplies

Below, you find the abbreviations for the different sample materials. These abbreviations are used in this manual and on our submission forms.
You can use 'MyLab' or order forms to order shipping materials. There you will also find the order numbers of the individual shipping materials.

A. **EB = EDTA blood**

B. **EP = EDTA plasma**
EDTA blood has to be centrifuged and the supernatant needs to be transferred into a neutral test tube (e.g. Eppendorf tube).

C. **HB = heparin blood**

D. **HP = heparin plasma**
Heparin blood has to be centrifuged and the supernatant needs to be transferred into a neutral test tube (e.g. Eppendorf tube).

E. **NaFB = sodium fluoride blood**

F. **NaFP = sodium fluoride plasma**
NaF blood has to be centrifuged and the supernatant needs to be transferred into a neutral test tube (e.g. Eppendorf tube).

G. **S = serum**
The coagulated blood should be centrifuged and the supernatant serum should be transferred into a neutral test tube or another serum tube (remove beads before!).

H. **CB = citrate blood**

I. **CP = citrate plasma**
The sample should be centrifuged and the supernatant should be transferred into an uncoated test tube (e.g. Eppendorf tube).

J. **Smear**
Smears should always be sent in air-dried, unfixed and unstained. For transportation, the depicted transport covers (shipping containers) are suitable.

K. **Shipping container**
for blood tubes or urine tubes

L. **Swab with transport medium**
(orange: thin swab, amies medium clear; black: thick swab, amies with charcoal)

M. **Swab without transport medium**

N. **Shipping container for swabs**

O. **Urine tube**

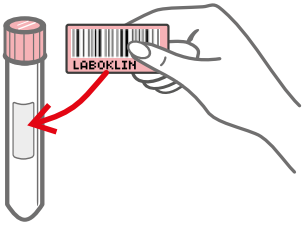
P. **Container for histology**
(formalin tube with shipping container)

Q. **Faeces tube**
with shipping container

R. **Blood culture flask set (aerobic and anaerobic)**

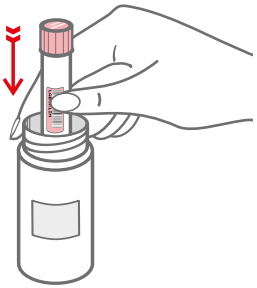
S. **Blood culture bottle Peds Plus™**

Labelling and shipment of samples



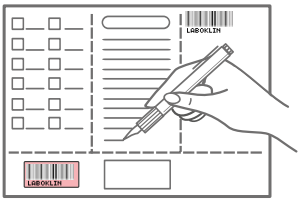
Step 1
Stick the bar code onto the test tube

Step 2
Fill the test tube

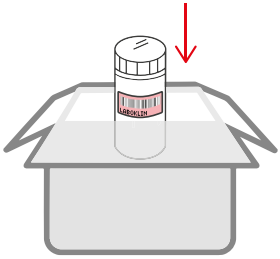


Step 3
Test tube into shipping container

Step 4
Additional bar code onto the shipping container



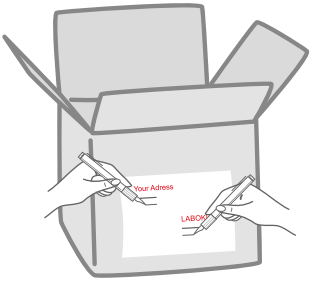
Step 5
Completely fill in submission form and, if necessary, sample list



Step 6
Put sample and submission form and, if necessary, sample list with sufficient cushioning material into the cardboard box

Step 7
Please choose the correct label and stick it on the box

Exempt Animal Specimen



Step 8
Pay attention to correct shipping information

2. Equine Profiles (clinical chemistry and haematology)

Our profiles are a combination of several clinical chemical and haematological parameters that allow for a quick overview of the horse's health status. The organ profiles are designed to confirm suspected diagnoses and for further monitoring of the patients.
Many profiles can be requested with an additional blood count, for which a reduced price is invoiced in these combinations.
Our profiles are subject to a certain "flow", as the included parameters are continuously checked to ensure they are up to date: with the ongoing progress in equine medicine, individual parameters are replaced, added or removed, so that LABOKLIN is able to always offer you profiles based on the latest scientific knowledge.
Information on our other horse profiles (PCR profiles, bacteriological/parasitological profiles, allergy profiles) can be found in the respective chapters.

2.1 Large Screening

Monitoring of all important organ functions as well as common minerals and trace elements

Parameter: AP, γ-GT, GLDH, total bilirubin, cholesterol, triglycerides, glucose, AST (GOT), LDH, CK, protein, albumin, globulins, albumin/globulin ratio urea, creatinine, PO4, Ca, Mg, K, Cl, Na, Fe, Zn, Cu, Se

Material: S 1 ml + NaFB 1 ml

Results: day of sample receipt (Mon – Sat; Se possibly the next working day)

Method: photometry, AAS

2.2 Large Screening + SAA

Parameter: like 2.1 + SAA

Material: S 1 ml + NaFB 1 ml

Results: day of sample receipt (Mon – Sat; Se possibly the next working day)

Method: photometry, AAS

2.3 Small Screening

Cost-effective starter profile for liver, kidney, muscles and metabolism

Parameter: GLDH, γ-GT, triglycerides, AST (GOT), LDH, CK, urea, creatinine, protein

Material: S 1 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

2.4 Performance Profile

Focus on muscle metabolism, supplemented by the most important liver, kidney and metabolic parameters plus electrolytes

Parameter: AP, γ-GT, GLDH, total bilirubin, cholesterol, triglycerides, glucose, SAA, AST (GOT), LDH, CK, protein, albumin, globulins, albumin/globulin ratio, urea, creatinine, PO4, Ca, Mg, Na, K, Cl, Fe

Material: S 1 ml + NaFB 1 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

2.5 Senior Profile

This profile includes the most important liver, kidney and metabolic parameters as well as those electrolytes and trace minerals that are essential for older horses. Many parameters that indicate PPID are included; therefore, it is absolutely necessary to send in serum (centrifuged and pipetted off soon after sampling) and NaFB for accurate glucose results! However, only fasting glucose (only feed hay and straw) has any reliable informative value.

Parameter: γ-GT, GLDH, lipase (DGGR), total bilirubin, triglycerides, glucose, protein, albumin, globulins, albumin/globulin ratio, urea, creatinine, PO4, Ca, Zn, Se, SDMA

Material: S 1 ml + NaFB 1 ml

Results: day of sample receipt (Mon – Sat; Se possibly the next day)

Method: photometry, AAS

2.6 Foal Profile

Includes parameters important in foal medicine.

Parameter: triglycerides, urea, creatinine, protein, γ-GT, Na, Ca, Mg, PO4, serum protein electrophoresis

Material: S 1 ml

Results: day of sample receipt (serum protein electrophoresis not on Saturday)

Method: photometry and capillary electrophoresis

2.7 Liver

- Liver 1
A quick overview of the most important liver enzymes and bile acids

Parameter: AST (GOT), GLDH, γ-GT, bile acids

Material: S 0.5 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

- Liver 2
Additional parameters provide information on liver function and possible pathogenesis.

Parameter: GLDH, AST (GOT), AP, γ-GT, total bilirubin, cholesterol, urea, bile acids, protein, albumin, globulins, albumin/globulin ratio, glucose, Na, K, Cl

Material: S 1 ml + NaFB 1 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

2.8 Kidney Large Animals

Parameter: urea, creatinine, protein, albumin, globulins, albumin/globulin ratio, Ca, PO4

Material: S 0.5 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

2.9 Muscular Screening

This profile is useful for diagnosis and follow-up, especially in myopathies. It includes the most important muscle enzymes plus electrolytes and minerals.

Parameter: CK, AST (GOT), LDH, Na, K, Cl, Ca, PO4, Mg, Fe

Material: S 1 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

2.10 Muscular Screening Extended

Parameter: CK, AST (GOT), LDH, Na, K, Cl, Ca, PO4, Mg, Fe, vitamin E, Se

Material: S 3 ml

Results: clinical chemical parameters on day of sample receipt (Mon – Sat), vitamin E after approx. 3 working days after sample receipt

Method: photometry, HPLC, AAS

2.11 PPID Profile (Equine Cushing)

This profile is designed to provide as much information as possible from a single fasted blood sample. Aetiological parameters are determined as well as indicative parameters. At the same time, insulin dysregulation is also examined.

Parameter: insulin, ACTH, glucose, fructosamines, triglycerides, γ-GT, RISQI, I/G ratio, MIRG (for proxies see EMS Profile)

Material: non-haemolysed EP 2 ml (cooled) + non-haemolysed S 2 ml (cooled) + NaFB 2 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry, CLIA

For further information concerning diagnostics of PPID see Chapter 8.5, page 71.

2.12 EMS Profile (Equine Metabolic Syndrome)

Apart from fasting insulin and glucose, further parameters are determined to assess the metabolic status of the horse.

Parameter: insulin, glucose, fructosamines, RISQI, I/G ratio, possibly MIRG

Note:
Fructosamines reflect the average blood glucose level of the past 2 to 3 weeks. RISQI (= reciprocal inverse square of insulin) is a "proxy", i.e. a mere calculation of insulin and glucose and represents a measure for insulin sensitivity. The proxies "insulin/glucose ratio" and MIRG (= modified insulin to glucose ratio) (only stated in borderline cases) reflect pancreatic β-cell function.

Material: S 2 ml (non-haemolysed, cooled) + NaFB 2 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry, CLIA

For further information concerning EMS diagnostics see Chapter 8.6, page 73.

2.13 Complete Blood Count

Includes erythrocytes, haemoglobin, haematocrit, platelets and leukocytes as well as a differential blood count. Depending on the clinical issue, the complete blood count can be ordered together with each profile. In addition to automated analysis, all samples with evident morphological abnormalities are also evaluated microscopically.

Material: EB 1 ml + blood smear

Results: day of sample receipt (Mon – Sat)

Method: laser light – possibly microscopy

2.14 Coagulation

This profile is for the diagnosis of rare coagulation disorders in horses. It can also be used for monitoring thrombolysis treatments (Quick). Fibrinogen can also be interpreted as an acute phase protein.

Parameter: prothrombin time (PT, Quick), PTT, thrombin time and fibrinogen

Material: CP 1 ml (cooled)

Results: day of sample receipt (Mon – Sat)

Method: chronometry

Cave:
A correct mix ratio of citrate and blood is extremely important – please observe the indicated fill level! In addition, the expiry date of the citrate tubes should be strictly adhered to.

2.15 Immune Status

The cellular immune status includes a complete blood count and the determination of B cells (CD21+), T cells (CD3+, CD5+), T helper cells (CD4+) and cytotoxic T cells (CD8+). In horses, it is used to clarify immunodeficiency in the case of frequent and prolonged infections.

Material: EB, HB 3 ml + blood smear – not older than 48 hours on arrival at the laboratory in Bad Kissingen!

Results: day of sample receipt (Mon – Fri)

Method: flow cytometry

2.16 Blood Donation Profile

Based on the German guidelines for the collection, storage, transport and administration of blood and blood products in the veterinary sector.

Parameter: complete blood count
urea, creatinine, Na, K, Ca, PO4, bilirubin, ALT, AP, AST, GLDH, protein, albumin, glucose, fibrinogen, equine infectious anaemia virus (Coggins test), equine arteritis virus (VNT), babesia (cELISA), urinalysis

Material: S 2 ml + EB 2 ml + NaFB 2 ml + CP 2 ml + urine 2 ml

Results: 3 working days after sample receipt

Method: laser light – possibly microscopy, photometry, dry chemistry, serology as stated

2.17 Mineral and Trace Elements Profile (Mineral Profile II)

Includes all important parameters of mineral metabolism and trace elements.

Parameter: Ca, Na, PO4, Mg, Zn, Cu, Se, K, Cl, Fe, Mn

Material: non-haemolysed S 3 ml

Results: 1 – 3 working days after sample receipt

Method: AAS, photometry, ICPMS

2.18 Iron Metabolism

The new profile allows you to differentiate between absolute (blood loss) and functional iron deficiency (sequestration due to inflammation, tumour or infection). Total serum iron, relative iron saturation as well as total and unsaturated iron binding capacity of the serum are determined. In addition, hepcidin as a regulatory hormone of iron metabolism and serum ferritin as a parameter of the iron stores of the organism.

Material: iron, ferritin, hepcidin, iron saturation, UIBC, TIBC, SAA

Material: S 0.5 ml (cooled)

Results: 2 – 5 working days after sample receipt

Method: photometry, LCMS

2.19 Heavy Metal Screening

If heavy metal poisoning is suspected.

Parameter: arsenic, lead, cadmium, chromium, copper, manganese, mercury, thallium, zinc

Material: S 2 ml + EB 2 ml + urine 5 ml

Results: 7 working days after sample receipt

Method: photometry, AAS, ICPMS

2.20 Neurology Profile

Parameter: serum amyloid A (SAA), antibodies: Flavivirus IgG, West Nile Virus IgM, tick-borne encephalitis virus (TBEV) (IgG and IgM), PCR: bornavirus, EHV 1 & 4

Material: S 1 ml + EB/nasal swab/(CSF)

Results: 2 – 4 working days after sample receipt

The probability of detecting herpes virus is increased if both EDTA blood and a nasal swab are submitted in addition to serum.

2.21 Tumour Diagnostics

Thymidine kinase, complemented by serum amyloid A, calcium and serum protein electrophoresis (for detailed parameter description see Chapter 3.5, page 29 and Chapter 3.6, page 30)

Parameter: thymidine kinase, SAA, Ca, serum protein electrophoresis

Material: S 1 ml (cooled)

Results: day of sample receipt (Mon – Fri)

3. Specific (Single) Parameters

3.1 Foal IgG

Insufficient supply of maternal immunoglobulins through the colostrum is one of the most predisposing factors for infectious diseases in foals. IgG determination in the blood of newborn foals by capillary electrophoresis provides an exact measurement of IgGs and allows for early diagnosis – before clinical symptoms occur – and initiation of therapeutic measures.

Material: S 0.5 ml

Result: day of sample receipt (Mon – Sat)

Method: capillary electrophoresis

3.2 Lactate

Is the end product of anaerobic glycolysis in muscle tissue. Determination of lactate concentration may be helpful for the diagnosis of myopathies or – within the scope of an exercise stress test – be used to check the individual fitness level of a horse.

Procedure:
- Blood collection before exercise = baseline value
- After some warm-up laps, physical exercise: approx. 10 min at 5 m/s (equivalent to canter)
- 2nd blood collection 3 min after exercise
- 3rd blood collection 15 min after exercise
- 4th blood collection 30 min after exercise, possibly for CK determination

Interpretation:
- Baseline value < 2 mmol/l
- 3 min value: not more than twice the baseline value
- 15 min value: 30 % lower than 3 min value

It may be useful to submit non-haemolysed serum from the baseline blood sample and the 30-minute value so that other muscle enzymes can be monitored, too.

Material: NaFB or NaFP 0.5 ml in each case

Results: day of sample receipt (Mon – Sat)

Method: photometry

It may make sense to send additional not-haemolysed serum from the basal blood sample and the 30-minute value in order to check other muscle enzymes as well.

3.3 DGGR Lipase

DGGR lipase is a specific and sensitive biomarker for the diagnosis of pancreatitis. Pancreatitis can be associated with colic or other gastrointestinal disease. Elevated DGGR lipase values can also be seen in horses with high intensity training.

Material: S, (HP, EP) 0.5 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

3.4 Symmetric Dimethylarginine (SDMA)

SDMA is an end product of the protein metabolism and – for the most part – excreted via the kidneys. SDMA serum concentrations reflect glomerular filtration rate and there is a good correlation between SDMA and creatinine. In contrast to creatinine, SDMA is affected little by muscle mass or -wasting. SDMA can be used for early detection of renal disease.

Material: S, HP 0.5 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

3.5 Serum Amyloid A (SAA)

SAA is considered a "major APP" (acute-phase protein) in horses and can be used to detect subclinical diseases or for monitoring of treatment/healing processes.

Material: S 0.5 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

3.6 Thymidine Kinase (TK-1)

Thymidine kinase can be used as a biomarker for lymphoma diagnosis in horses. Horses with lymphoma show significantly higher activity of thymidine kinase than control groups.

Material: S 0.5 ml (cooled)

Results: day of sample receipt (Mon – Sat)

Method: CLIA

3.7 Alpha Fetoprotein (AFP)

AFP can be used as a tumour marker; especially in case of liver tumours values may be elevated.

Material: S 1 ml

Results: day of sample receipt (Mon – Sat)

Method: CLIA

3.8 Iodine/Creatinine Ratio

The iodine supply can be assessed very well by the determination of the iodine excretion via the urine. The correlation between alimentary iodine intake and renal iodine excretion is very good.

Material: urine

Results: 3 – 4 working days after sample receipt

Method: ICPMS, photometry

3.9 Manganese

Manganese acts as an essential trace element and is involved in several metabolic processes, such as bone and cartilage formation, and also has an antioxidant effect. A deficiency of manganese can lead to various health problems, including skeletal abnormalities, reduced fertility and lower resistance to stress.

Material: S 0.5 ml

Results: 2 – 3 working days after sample receipt

Method: AAS

3.10 Vitamin E

Vitamin E is a so-called radical scavenger that can metabolise free radicals and thus neutralise them. Vitamin E is therefore particularly important for the muscles and heart. The supply of vitamin E is often too low, especially during physical exertion or in the winter months.

Material: S 0.5 ml (cooled)

Results: 3 working days after sample receipt

Method: HPLC

Reference values:
>2 mg/l: adequate supply
1.5-2 mg/l: marginal supply
<1.5 mg/l: deficient supply

4. Infectious Diseases

4.1 Bacteria
4.1.1 Borrelia
4.1.2 Clostridioides difficile: Toxin A and B
4.1.3 Clostridium perfringens: Alphatoxin, Enterotoxin, netE and netF
4.1.4 Lawsonia intracellularis
4.1.5 Leptospira
4.1.6 Listeria
4.1.7 Rhodococcus hoagii (formerly R. equi)
4.1.8 Salmonella
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4.2 Viruses
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4.2.2 Equine Arteritis Virus (EAV) (Equine Viral Arteritis, EVA)
4.2.3 Equine Coronavirus
4.2.4 Equine Hepacivirus (EqHV)
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4.2.5.1 Equine Herpesviruses 1 & 4 (EHV1 & EHV4)
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4.2.8 Influenza A Virus
4.2.9 Papillomaviruses
4.2.9.1 Bovine Papillomaviruses 1 & 2 (BPV 1/2, Equine Sarcoid)
4.2.9.2 Equine Papillomavirus (EcPV)
4.2.10 Rotavirus A
4.2.11 Tick-borne Encephalitis Virus (TBEV)
4.2.12 West Nile Virus

4.3 Blood Parasites
4.3.1 Anaplasma
4.3.2 Theileria equi/Babesia caballi – Babesiosis – Piroplasmosis

4.4 Endoparasites
4.4.1 Strongylids
4.4.2 Larval Cyathostominosis (Small Redworm)
4.4.3 Roundworms (Parascaris equorum, P. univalens)

There are 2 different ways to approach the diagnosis of infectious diseases:
1) direct detection of the pathogen by culture or PCR or
2) indirect serological examination that reveals previous contact with the pathogen by detection of antibodies
The clinical relevance of antibody titres often can only be assessed by looking at the titre curve. Epidemiological factors have to be considered as well. In equine medicine, the examination of paired titres, for example, is common in many clinical issues when it is not only a matter of the antibody concentration but also especially of fall or rise in titre.
It should also be taken into account that significant IgG titres can only be measured from about 3 weeks onwards after initial contact with the pathogen.

4.1 Bacteria

4.1.1 Borrelia

In Europe, this tick-borne disease is presumably caused by 3 pathogenic Borrelia species of the group Borrelia burgdorferi sensu lato: B. burgdorferi sensu strictu, B. afzelii, B. garinii.
Although the suspected diagnosis of Lyme borreliosis is becoming increasingly common in practice, the diagnosis of the disease is still difficult. In endemic areas, serological prevalence is high even in healthy horses. Numerous clinical findings in horses are

associated with Borrelia: performance reduction, chronic intermittent or alternating lameness, alterations of the skin, eyes or heart up to neurological deficits and abortions. Whether or not this spirochete infection causes any clinical symptoms in horses at all is still being controversially discussed.

Detection:
- DNA detection in skin biopsies, CSF, synovia or in ticks by PCR
- quantitative antibody detection: IgM and IgG by IFAT in serum or EP, HP
- qualitative antibody detection: Borrelia blot: Borrelia IgG Line immunoassay in serum (suspicious titres should always be confirmed by Line immunoassay)

Results:
PCR: 1 – 3 working days after sample receipt
IFAT: day of sample receipt (Mon – Fri)
blot: 3 working days after sample receipt

4.1.2 Clostridioides difficile Toxin A and B

Clostridia are obligate anaerobic spore formers. In small quantities and without toxin production, they are part of the physiological intestinal flora. Detection of toxins / toxin genes is therefore essential for diagnosis.
Various predisposing factors play a role in triggering strong proliferation and clinical disease caused by C. difficile: antibiotics, hospitalisation, dysbiosis, etc. In horses, C. difficile is associated with necrotising colitis in foals, post-antibiotic colitis in adults, and the "duodenitis-proximal jejunitis syndrome." The signs are often non-specific (diarrhoea, colic, fever).

Detection:
- PCR from faeces (detection of the toxin gene)
- ELISA from faeces (detection of the toxin)

Results:
PCR: 1 – 3 working days after sample receipt
ELISA: 1 – 2 working days after sample receipt

Detection by PCR is also part of the Large Faecal Profile, the Foal Faecal Profile and the profile Foal Diarrhoea Pathogens.

4.1.3 Clostridium perfringens: Alpha Toxin, Enterotoxin, netE and netF Toxin

Clostridia are obligate anaerobic spore-forming organisms. They are part of the physiological intestinal flora and are also present in the environment. Detection of toxins /toxin genes is therefore essential for diagnosis. Clostridium perfringens is classified into toxovars based on the presence of various toxins. Various predisposing factors, especially all types of dysbiosis, lead to rapid proliferation and clinical disease. The alpha toxin is produced by all toxin-producing C. perfringens and is a microbial exotoxin. The enterotoxin binds to the intestinal wall and forms pore-like structures there, which lead to destruction of the cell wall and cell death. NetE and netF are also pore-forming toxins.

Detection: alpha toxin: PCR from faeces (detection of the toxin gene)
enterotoxin: - PCR from faeces (detection of the toxin gene)
- ELISA from faeces (detection of the toxin)
netE toxin: PCR from faeces (detection of the toxin gene)
netF toxin: PCR from faeces (detection of the toxin gene)

Results: PCR: 1 – 3 working days after sample receipt
ELISA: 1 – 2 working days after sample receipt

Detection by PCR is also part of the Large Faecal Profile (enterotoxin) and the Foal Faecal Profile (enterotoxin and netF) as well as the profile Foal Diarrhoea Pathogens (netF).

4.1.4 Lawsonia intracellularis

Over the past years, this obligate intracellular, gram-negative bacterium has proven to be an important pathogen in the differential diagnosis of diarrhoea in foals. Especially weanlings (< 6 – 7 months of age) are affected, in which L. intracellularis colonises the crypt cells of the ileum causing proliferative enteropathy. This may result in intestinal malabsorption and/or (mostly) chronic diarrhoea. In most cases, individual animals fall ill, though multiple illnesses have been described in some farms. Clear findings in abdominal ultrasound and hypoalbuminaemia are indicative of Lawsonia infection.

Detection: since only very few pathogens are excreted with the faeces, detection by PCR is the method of choice. However, false negative results may occur due to the very low excretion rate.

Results: 1 – 3 working days after sample receipt

4.1.5 Leptospira

Leptospira infections, which are spread through the urine of rodents, are usually clinically inapparent in horses. Thus, the seroprevalence in healthy horses is high. The pathogen is ingested with feed or water and leads to rather non-specific symptoms in horses, like fever (often intermittent), icterus, inappetence and productivity loss. Abortions have been described as well. Transmission of the pathogen between horses does practically not occur.

Special case: equine recurrent uveitis (ERU)
Intraocular persistent leptospira infections seem to contribute to the aetiology of ERU. Resulting autoimmune reactions lead to a progressive deterioration of the inner structures of the eye and may even lead to blindness. Detection of antibodies (= most sensitive test) or pathogen detection (using PCR) in aqueous humour or tissue of the vitreous body indicate ERU.

Cave: serum antibody titres are irrelevant in ERU!

Detection:
- DNA detection by PCR in EB (acute febrile illness) or urine (chronic disease) or aqueous humour or tissue of vitreous body (ERU)
- quantitative antibody detection: MAT for serovars:
  L. interrogans Australis, Autumnalis, Bratislava, Canicola, Grippotyphosa, Icterohaemorrhagiae, Pomona, Saxkoebing, Sejroe
  in serum (systemic disease)
  or in aqueous humour/tissue of vitreous body (with ERU signs)

Results:
PCR: 1 – 3 working days after sample receipt
MAT: day of sample receipt (Mon – Fri)

During acute systemic disease, a significant rise in titres of one or more serovars is to be expected.

4.1.6 Listeria

Listeria are ubiquitous pathogens with a wide range of hosts. They can also be found in the intestine of healthy animals. In horses, only L. monocytogenes plays a role. Transmission between horses is possible, but it is more likely that the pathogen is taken up from the environment, e.g. via contaminated feed. Listeria particularly multiply in silage. Clinical manifestation as septicaemia, encephalitis or abortion is rare. Listeriosis is a notifiable disease!

Detection:
- quantitative antibody detection by IFAT
- culture from CSF, abortion material, etc. is possible but requires special culture media; it is therefore essential that suspected listeriosis is noted on the submission form.
- PCR from abortion material

Results:
IFAT: day of sample receipt (Mon – Fri)
culture: 1 – 2 working days after sample receipt
PCR: 1 – 3 working days after sample receipt

Because of the widespread distribution of the pathogen, a significant rise in titre for 2 – 3 weeks in conjunction with acute symptoms would indicate listeriosis.

4.1.7 Rhodococcus hoagii (formerly: R. equi)

Rhodococcus hoagii causes severe pneumonia in foals. Infection occurs during the first days of life by inhalation of pathogens bound to dust particles. The course of the disease is gradual. At the earliest, clinical signs occur at the age of 3 – 4 weeks, sometimes even after several months. Purulent bronchopneumonia and abscess formation in the lungs occur. When the pathogens are swallowed, they reach the gastrointestinal tract where they proliferate and may cause granuloma and diarrhoea. They are excreted with the faeces and thus released into the environment – a source of infection for other foals. Older animals also excrete the pathogen but do not fall ill themselves. Moreover, R. hoagii shows an affinity for bones and joints.

Detection:
- detection from tracheobronchial secretions (TBS), faeces or nasal swabs.
- bacterial culture: TBS and faeces are preferred (higher sensitivity).
- PCR: this highly sensitive method can help to identify clinically healthy shedders.

Results: culture: 1 – 2 working days after sample receipt
PCR: 1 – 3 working days after sample receipt

4.1.8 Salmonella

Salmonella spp. belong to the family Enterobacteriaceae and are found in the intestines of animals and humans. Combined with predisposing factors like immunodeficiency or stress, salmonellosis in horses can lead to diarrhoea and fever, septicaemic forms often occur in young animals.
Asymptomatic „carriers" are described as well, they do not get sick but are a source of infection for other animals and humans.
In Germany, Salmonellosis in horses is a reportable disease.

Detection:
- bacterial culture (also included in the Large and Small Faecel Profil and in the Foal Faecel Profile)
- by PCR from faeces

Results: culture: 2 – 3 working days after sample receipt
PCR: 1 – 3 working days after sample receipt

4.1.9 Staphylococci with Methicillin Resistance

Methicillin-resistant staphylococci are regularly detected in horses. MRSA (methicillin-resistant Staphylococcus aureus) can often be isolated from wounds, for example.
If staphylococci are detected in a bacterial culture, further screening for methicillin resistance is carried out using a special culture medium. Methicillin resistance can also be detected using a special antibiogram or PCR (detection of the mecA/mecC gene). PCR is also only possible after prior cultivation.

Detection: bacteriological culture from swab with transport medium
+ special culture medium (at no additional cost)
or + PCR
or + specially developed antibiogram (included in the profile Analysis on Multidrug Resistant Bacteria)

Results: culture + special culture medium: 2 – 3 working days after sample receipt
culture + PCR: 3 – 6 working days after sample receipt
culture + special antibiogram: 3 – 4 working days after sample receipt

4.1.10 Streptococcus equi equi (Strangles) / Streptococcus equi zooepidemicus

The globally spread equine disease strangles is caused by an infection with **Streptococcus equi subspec. equi.** The main clinical signs are purulent infections of the lymph nodes of the head. Further symptoms are: high fever, nasal discharge, coughing and lethargy. Strangles is a highly contagious disease, transmission is not only possible by direct contact but also indirectly (e.g. objects). Therefore, well-coordinated herd management is mandatory. The problem is: approx. 2 – 10 % of the affected animals become carriers, i.e. the pathogen is not completely eliminated but retreats into the guttural pouch and is excreted intermittently. Carriers are often asymptomatic and therefore easily overlooked. However, identification of carriers is absolutely necessary to avoid a permanent manifestation in the herd and an outward spread of the disease. PCR is more sensitive than bacterial culture but as it cannot distinguish between live and dead organisms, it remains unclear whether or not an animal is infectious. Thus, a combination of both methods is recommended.
Clinically, an infection with Streptococcus equi subsp. equi cannot always be distinguished from an infection with **Streptococcus equi subsp. zooepidemicus**, a facultative pathogenic commensal in horses. Infections can cause respiratory disorders and purulent bronchopneumonia.

Detection:
- material: guttural pouch lavage! (gold standard, highest sensitivity), pharyngeal flush, aspirate (lymph node), TBS, BAL, swab (nose)
- PCR: swab without medium; it can be chosen between the single detection of Streptococcus equi equi or the combined detection of Streptococcus equi equi and Streptococcus equi zooepidemicus
- bacteriological examination: swab with medium
- antibody detection by ELISA (serum or plasma).

The ELISA used at Laboklin detects antibodies against the highly specific proteins A and C of Streptococcus equi subsp. equi. The test enables the detection of both symptomatic and asymptomatic (carrier) horses that tend to excrete the bacteria. It can be used for the following purposes:
- detection of a recent infection, after only 2 weeks
- identification of exposed animals without clinical signs

4.1.11 Taylorella

Taylorella equigenitalis is the causative agent of contagious equine metritis (**CEM**). Information on diagnostics, which is mainly carried out as part of export tests, can be found in Chapter 5.6, page 55.
Taylorella asinigenitalis is mostly apathogenic, but pathogenic strains can lead to severe purulent endometridia, especially in horses. Testing is recommended as part of breeding hygiene. For further information, see Chapter 6.4.3, page 60.

4.1.12 Tetanus – Vaccination Titre

The detection of antibodies against Clostridium tetani toxin is particularly useful for determining the immunisation of the horse.

Detection: antibody detection by ELISA in serum

Results: day of sample receipt (Mon – Fri)

Analysis is semi-quantitative:
- reliable immunisation provided
- or: immunisation provided
- or: no tetanus antibodies can be detected.

4.2 Viruses

4.2.1 Borna Disease Virus

In horses, Borna disease virus (BDV) causes non-purulent encephalomyelitis; BVD can be found in a variety of species and leads to neurological signs as well as behavioural disorders. Horses and sheep seem to be the most susceptible species. Infected horses are mainly found in East Germany and in Switzerland. However, epidemiological studies show a seroprevalence of approx. 11.5 % in all federal states of Germany. In horses from "Borna livestocks", seropositivity increased to 33 %. In general, asymptomatic infections and clinical cases must also be expected outside the endemic areas. Shrews were identified as virus reservoir. These are asymptomatic but infected for life. Other mammals such as horses and sheep as well as humans can act as false hosts. In humans, infections are very rare individual cases. Encephalitis caused by the virus is usually fatal.

Detection:
- quantitative antibody detection by IFAT from serum
- detection of antibodies in CSF would also confirm the diagnosis
- pathogen detection by PCR in CSF, EB (viraemia), intraocular fluid, retina, brain tissue

Results:
IFAT: 7 working days after sample receipt
PCR: 1 – 3 working days after sample receipt

In Germany, borna is a notifiable disease!

4.2.2 Equine Arteritis Virus (EAV) (Equine Viral Arteritis, EVA)

Equine Viral Arteritis (EVA) is a worldwide distributed, contagious viral infection of equids caused by the equine arteritis virus (EAV).
Confirmed outbreaks seem to have increased in recent years. The majority of naturally acquired infections is subclinical, nevertheless, seroconversion still occurs.

When clinical signs appear, they vary in type and severity: fever, depression, anorexia and peripheral oedema, conjunctivitis ("pink eye"), urticaria and abortion. In young animals, fulminant pneumonia and pneumoenteritis may also be seen.
The virus is mainly transmitted through ejaculate. Persistently infected carrier stallions carry the virus in their accessory sex glands and intermittently shed it in the genital secretions. Geldings, prepubescent stallions and mares cannot be carriers.
Especially in animals with systemic disease, virus shedding can also occur by other body secretions: aerosolised secretions of the respiratory tract, urine, abortion material, etc.

Detection:
- detection of RNA by PCR from stallion semen/ejaculate, in systemic affected animals from secretions of the respiratory tract, urine, abortion material, etc.
- during viraemia (fever!), PCR can be attempted from EB.
- quantitative antibody detection by VNT; if titres are low or borderline, a second testing is recommended after 3 to 4 weeks. Vaccination titres cannot be distinguished from infection titres.

Results: PCR: 1 – 3 working days after sample receipt
VNT: 5 working days after sample receipt

Cave: in Germany, EVA is a reportable disease (horses, donkeys, etc.).

4.2.3 Equine Coronavirus (ECoV)

Equine coronavirus (ECoV) has recently been detected in the USA, Japan and Europe and is associated with fever, colic and diarrhoea. Particularly adult horses are affected in the cold season. Neurological abnormalities have rarely been described: secondarily caused by hyperammonaemia.
Infections can affect several animals of a herd but seem to be self-limiting. However, secondary complications can worsen the course of the disease. Transmission occurs mainly via the faecal-oral route.

Detection: PCR from faeces

Results: 1 – 3 working days after sample receipt

4.2.4 Equine Hepacivirus (EqHV)

Hepaciviruses belong to the Flaviviridae. The equine hepacivirus was previously known as non-primate hepacivirus (NPHV). Transmission occurs primarily via blood products (plasma, tetanus anti-toxin, PMSG, ...) and iatrogenically, as well as vertically. The virus is hepatotropic and is associated with liver disease (hepatitis). However, an infection can also be subclinical.

Detection: DNA detection by PCR from EDTA blood, serum, liver tissue (also embedded in paraffin)

Results: 1 – 3 working days after sample receipt

4.2.5 Equine Herpesviruses

4.2.5.1 Equine Herpesviruses 1 & 4 (EHV1 & EHV4)

Infections with EHV1 as well as with EHV4 primarily cause diseases of the respiratory tract. The severity of the clinical symptoms depends on the age and immune status of the infected animal.
Particularly infections with EHV1 are able to spread beyond the respiratory mucosa and cause severe manifestations of the disease: abortions, perinatal foal death, neurological diseases.
In foals infected with EHV4, morbidity rates of up to 100 % are possible, especially during weaning from the dam. More than 80 % of isolates come from animals with rhinopneumonitis.
Once infected, horses remain carriers of the virus for life. The virus can be reactivated endogenously under adverse conditions (stress, etc.).
Lymph organs, the leukocyte fraction and trigeminal ganglia are the main latency organs. If the vaccinated horses are also taken into account, seroprevalence in the horse population is high.
In recent years, EHV-1-associated neurological diseases, for which a "neurotropic" strain of EHV1 is held responsible, have been reported with increasing frequency and severity of the clinical disease. This much-feared clinical picture is referred to as EHM (equine herpesvirus myeloencephalopathy).
In horses, 2 different variants (DNApol D752 vs. DNApol N752) with varying neuropathogenicity have been described. The D752 variant is associated with most of the neurological disease outbreaks and is therefore referred to as neuropathogenic. However, only some of the infected horses develop neurological signs. The N752 variant is mainly found in case of abortions, but also in a smaller number of neurological diseases. Differentiation is particularly interesting from the epidemiological point of view.

Detection:
- PCR from nasal swabs/secretions of the respiratory tract, CSF or abortion material including foetal membranes or aqueous humour.
  Recent studies suggest examining a sample of EDTA blood in parallel with the organ material. This is supposed to increase the probability of detection.
  In case of a positive EHV1 result by PCR, the differentiation of the EHV1 variant is performed automatically and free of charge.
  The detection of EHV1 or EHV4 can be ordered as a single test. The detection of EHV1 and EHV4 is included in the different respiratory profiles (see Chapter 4.5.8, page 52.). Furthermore, the detection of EHV1 and/or EHV4 by PCR is part of the following profiles: Neurology (see Chapter 2.20, page 27), Abortion (see Chapter 4.5.1, page 51) and Uveitis (see Chapter 4.5.5, page 51).
- antibody detection by ELISA which can sensitively distinguish between EHV1 and EHV4 antibodies.

Results:
PCR: 1 – 3 working days after sample receipt
ELISA: 2 – 3 working days after sample receipt

A clear increase in titre of paired serum samples (interval 10 – 14 days) indicates an acute EHV infection. Vaccination titres cannot be distinguished from infection titres! In acute cases of illness, however, we recommend direct pathogen detection by PCR.

4.2.5.2 Equine Herpesviruses 2 & 5 (EHV2 & EHV5)

The involvement of EHV2 and/or EHV5 in keratoconjunctivitis has long been suspected and these viruses are indeed regularly detected in conjunctival swabs. In recent years, it has increasingly been shown that EHV2 and 5 are precursors of other viral and bacterial infections of the respiratory tract. Especially in young animals, EHV2 and/or 5 were detected in treatment-resistant, partly catarrhal-purulent, partly necrotising or abscessing bronchopneumonia. EHV5 was presented as aetiological agent of "equine multi-nodular pulmonary fibrosis" (EMPF).

Detection:
- using PCR
- EHV2 and EHV5: conjunctival swab (ideally taken with cytobrush), aqueous humour, cornea, EDTA blood
- EHV5 for suspected EMPF: tracheobronchial secretion (TBS), bronchoalveolar lavage (BAL), lung tissue
- the detection of EHV2 or EHV5 can be ordered as a single test, the profile Eyes includes the detection of both pathogens.

Results: 1 – 3 working days after sample receipt

4.2.5.3 Equine Herpesvirus 3 (EHV3)

Coital exanthema caused by EHV3, which only sporadically occurs in Germany, is a mildly progressing venereal infection in horses. Clinically, blisters, pustules and erosions appear on the mucous membrane of the vestibule, penis or prepuce as well as on adjacent skin areas. Healing takes place spontaneously after approximately 2 – 3 weeks, but can be complicated by secondary infections. Coital exanthema is a typical venereal infection. However, transmission is also possible through close contact as well as rectal and vaginal examinations. Infected animals remain carriers of the virus for life.

Detection: PCR from swab without medium (lesions on vestibule, penis, prepuce or surrounding skin) or tissue (lesions)

Results: 1 – 3 working days after sample receipt

4.2.6 Equine Infectious Anaemia Virus (EIAV)

Equine infectious anaemia (EIA) is a worldwide distributed disease in equids with acute lethal to chronic recurrent forms. EIA is characterised by recurrent fever, anaemia, thrombocytopenia, oedema and considerable weight loss.
Transmission takes place via infected blood, blood-sucking insects, iatrogenic through infected injection equipment, but also intrauterine.

Once infected horses remain infectious and seropositive throughout their lives. All horses older than 6 months that are seropositive are thus considered carriers; younger horses can be seropositive through maternal antibodies. Normally, the incubation period is 1 – 3 weeks, but may also last up to 3 months.

Detection: first antibodies can be detected 2 – 3 weeks post infection. If the results of the serological examination are negative but animals are suspected of being infected, the test should be repeated within 3 – 4 weeks.

Method:
- "Coggins test" (= agar gel diffusion test)
  To date, the Coggins test is the standard for all official procedures as well as for export tests.
- cELISA
  This test offers a considerably higher sensitivity than the "Coggins test"; that is why false positive results may occur in rare cases. However, these would only have implications if they were confirmed by a positive Coggins test.
  Negative cELISA results, in contrast, can be considered reliable.

Results: Coggins test: 3 working days (Mon – Sat) after sample receipt
cELISA: day of sample receipt (Mon – Fri)

Cave: EIA is an epizootic disease that is notifiable upon suspicion in many countries!

4.2.7 Equine Parvovirus (EqPV-H)

Equine serum hepatitis, also known as Theiler's disease, is caused by infection with equine parvovirus-hepatitis virus (EqPV-H). EqPV-H is a hepatotropic single-stranded and non-enveloped DNA virus. To date, two different modes of transmission have been assumed: One is the administration of products prepared from equine sera containing equine parvovirus. These include tetanus antitoxin, botulinum antitoxin, stem cell preparations, and equine plasma products in general. On the other hand, EqPV-H outbreaks also occur in horses that have not received a biological preparation in the past. In this case, transmission from horse to horse or spread by insects is assumed, but is currently still the focus of research.
It is presently thought that perhaps 2 % of infected horses develop clinical signs. Clinical signs of EqPV-H infection occur approximately 4 – 10 weeks after administration of a biological product contaminated with the virus. The spectrum of disease progression ranges from asymptomatic to fulminant liver failure. Acute hepatitis may present with neurologic signs, such as manic behavior, head pressing, and ataxia, in addition to lethargic behavior with associated anorexia. Colic, recumbancy or death within 72 hours have also been described.

Detection: DNA detection by PCR from EDTA blood or liver tissue (native)

Results: 1 – 3 working days after sample receipt

4.2.8 Influenza A Virus

Equine influenza is caused by the subtypes Influenza A equi 2 American and European type. In susceptible equids, an infection causes fever and a rough, dry cough. In unvaccinated populations, the virus spreads quickly. Secondary bacterial infections with mucopurulent nasal discharge are frequent and mask the clinical picture, especially in partially immune populations.

Detection:
- PCR from nasal swabs, TBS/BAL allows a quick and reliable diagnosis
- quantitative antibody detection by HAH: testing of paired serum at an interval of 10 – 14 days

Results:
PCR: 1 – 3 working days after sample receipt
HAH: 5 working days after sample receipt

Antibodies against A equi 2 American and European type are detected. A fourfold increase in titre would generally indicate an acute infection. Vaccination titres cannot be distinguished from infection titres.

4.2.9 Papillomaviruses

4.2.9.1 Bovine Papillomaviruses 1 & 2 (BPV 1/2, Equine Sarcoid)

The equine sarcoid is one of the most common skin tumours in horses (about 2 – 12 % of all horses are affected). The causative agent is bovine papillomavirus – especially type 1, more rarely type 2. The tumour cells are modified fibroblasts; the skin and subcutaneous tissue are affected. Equine sarcoids are considered semi-malignant tumours, i.e. they do not metastasise, but have a strong tendency to recur if surgical removal is incomplete. It is presumed that transmission mainly occurs through direct contact as well as flies and horseflies, but also indirectly through wound sites, saddles, blankets and cleaning utensils. The entire skin surface as well as certain blood cells are infected; moreover, the infection remains throughout life. The initial diagnosis is made at the age of 3 – 12 years.

Detection: pathogen detection by PCR from scurf, hairs (hair roots!) or tissue (tumour) samples. Positive PCR results confirm the suspected clinical diagnosis. Histopathology continues to be the gold standard for the diagnosis of equine sarcoid.

Results: 1 – 3 working days after sample receipt

4.2.9.2 Equine Papillomavirus (EcPV)

The equine papillomavirus (Equus caballus papillomavirus, EcPV) causes benign skin tumours in the genital area or on the ears ('aural plaques'). Different subtypes are responsible for this; 9 types have been described to date. In rare cases, aural plaques can develop into squamous cell carcinomas. Transmission probably occurs via direct contact.

Detection: virus detection using PCR from tissue

Results: 1 – 3 working days after sample receipt

4.2.10 Rotavirus A

Important pathogen causing diarrhea, especially in young animals. Transmission occurs via the faecal-oral route, the incubation period is 16 – 40 hours. Clinically, infection is characterised by sudden onset of watery diarrhoea, which is usually self-limiting. Secondary bacterial infections can lead to severe courses with dehydration and high mortality.

Detection:
- PCR form faeces
- antigen detection by ELISA from faeces

Results: PCR: 1 – 3 working days after sample receipt
ELISA: 1 – 2 working days after sample receipt

Caution: zoonotic potential!

4.2.11 Tick-borne Encephalitis Virus (TBEV)

Increasingly, TBE is also detected in neurologically affected horses. Like the West Nile virus, the TBE virus (TBEV) belongs to the genus Flavivirus. The clinical course of the disease is similar to that of West Nile virus disease (see Chapter 4.2.12).

Detection: ELISA: IgG and IgM in serum, IgG in CSF
PCR: pathogen detection in CSF, serum, possibly in tick

Results: ELISA: 2 – 3 working days after sample receipt
PCR: 1 – 3 working days after sample receipt

4.2.12 West Nile Virus

The virus is transmitted by blood-sucking insects.
Susceptible dead-end hosts are horses and humans; birds can become infected and serve as the virus reservoir, thus possibly transferring the virus over long distances. Infected horses show signs of encephalitis, but also ataxia, muscle tremor and paralysis can be found. About 10 % of infected horses develop neurological signs. About 30 % of these infected horses suffer a relapse after initial improvement of the signs, in which the lethality is then high (30 - 50 %). Infected horses are not infectious ('dead end' host).

Detection: ELISA: (Flaviviruses IgG und West Nile Virus IgM in horses, only Flaviviruses IgG in birds), required material: S 0.5 ml
RNA detection by PCR in EB, CSF, tissue (brain, spleen)

Results: ELISA: 2 – 3 working days after sample receipt
PCR: 1 – 3 working days after sample receipt

The very short viraemia phase (approx. 1 – 3 days) makes pathogen detection difficult. Furthermore, this phase is prior to the onset of the first clinical signs.

The flavivirus IgG ELISA detects a broad spectrum of flaviviruses such as West Nile virus, Usutu virus, TBE virus etc. due to the high cross-reactivity within the flavivirus antibodies. Positive IgG antibodies thus indicate flavivirus-specific antibodies. Further differentiation into antibodies specific for West Nile virus is not possible with the ELISA method. For positive IgM results, further differentiation using virus neutralisation tests (VNT) is therefore advisable. The detection of IgM antibodies is notifiable in many countries and indicates a recent infection.

4.3 Blood Parasites

4.3.1 Anaplasma

(formerly: "equine ehrlichiosis")
Anaplasma phagocytophilum (formerly: Ehrlichia equi), an obligate intracellular gram-negative, coccoid bacterium is the causative agent of equine granulocytic ehrlichiosis. In Europe, tick-borne granulocytic anaplasmosis is predominant. Inoculation and subsequent lymphogenic and haematogenous spreading are followed by colonisation of the target cells: neutrophil and eosinophilic granulocytes. Initial clinical signs include fever, apathy, limb oedemas and reluctance to move. Laboratory findings are thrombocytopenia and possibly mild anaemia. Animals > 4 years show more pronounced clinical signs than younger horses. Once the infection is overcome, horses acquire immunity which lasts for approximately 2 years.

Detection:
- DNA detection by PCR in EB of affected horses approx. 5 days after inoculation, i.e. at the same time as the onset of fever, whereas microscopic detection of blood parasites is not possible until about 5 days after the onset of the febrile illness.
- quantitative antibody detection by IFAT

Results:
PCR: 1 – 3 working days after sample receipt
microscopy: 1 – 2 working days after sample receipt
IFAT: day of sample receipt (Mon – Fri)

4.3.2 Theileria equi/Babesia caballi – Babesiosis – Piroplasmosis

Equine babesiosis or piroplasmosis is a tick-borne protozoal infection which is endemic in most tropic and subtropical territories and even reaches the temperate zones. Due to transportation of horses and expansion of the vector's distribution area, clinical cases and seropositive horses can now also be expected in Germany. Causative agents are Babesia caballi and Theileria equi (formerly Babesia equi) which can be found in the erythrocytes of infected animals. Clinical signs often are non-specific, the course of the disease is peracute to chronic. Characteristic are: fever – also intermittent –, anorexia, increased respiratory and cardiac rates, depression, anaemia, icterus and haemoglobinuria; in case of a chronic course of the disease: weight loss. Infected animals remain carriers for a long time and are therefore a source of infection for vectors.

Detection:
- PCR (Babesia spp./Piroplasmida) in EB. The first step is the detection of Babesia/Theileria. If the result is positive, differentiation between Theileria equi/Babesia caballi is performed subsequently by sequencing and free of charge.
- microscopic identification of pathogens in a blood smear
- quantitative antibody detection by
  - CFT (usually required for export-relevant tests)
  - cELISA (particularly for export to the USA, most sensitive test)
  - IFAT (only available for export examinations, if required)

Results:

PCR:	1 – 3 working days after sample receipt
microscopy:	1 – 2 working days after sample receipt
CFT:	5 working days after sample receipt
cELISA:	2 – 3 working days after sample receipt
IFAT:	day of sample receipt (Mon – Fri) (for export only)

4.4 Endoparasites

4.4.1 Strongylids

Most common endoparasite of the horse. All age groups are affected by infections. The strongylids can be divided into 2 subfamilies, the large and the small strongylids. Large strongylids are now only found on a few farms. Their larvae cause considerable damage during their body migration. Small strongylids can be found in every horse population.

Detection:
- flotation
- modified McMaster method when using selective deworming management
- egg count reduction test to assess the effectiveness of the anthelmintic used. For this purpose, the number of strongylid eggs is measured before and 14 – 21 days after deworming using the modif. McMaster method.

- larval culture to differentiate between large and small strongylids after previous strongylid detection. Please note: larval culture is only possible from fresh faeces. For reorders, faeces must therefore be sent in again.

Results: flotation, modif. McMaster method: day of sample receipt (Mon – Fri)
larval culture: 10 – 12 calendar days after sample receipt

4.4.2 Larval Cyathostominosis (Small Redworm)

Larval cyathostominosis is a disease caused by larvae of the small strongyles. From autumn until the end of winter, the number of larvae encapsulated in the intestinal mucosa increases considerably. The larvae remain in this dormant state until spring. At the beginning of spring, there is a synchronised mass migration of the larvae into the intestine, which leads to severe damage to the intestinal mucosa, which is literally 'perforated.' Young horses up to the age of 6 years are particularly affected.

Detection: antibody detection via ELISA from serum

Results: 5 – 7 working days after sample receipt

The test can still show a false positive result up to 4 months after successful deworming.

4.4.3 Roundworms (Parascaris equorum, P. univalens)

Signs such as diarrhoea, weight loss and reduced performance occur mainly in young animals, while adult horses often show a subclinical course. Complications include colic due to roundworm ileus or perforation of the small intestine. Roundworm eggs are extremely resistant. Observe hygiene!

Detection: flotation method

Results: day of sample receipt (Mon – Fri)

Caution:
The egg excretion does not correlate with the worm burden.
A postive result therefore always indicates deworming.

4.4.4 Tapeworms: Anoplocephala perfoliata, A. magna, Paranoplocephala mamillana

Infections occur through the ingestion of moss mites containing cysticerci with the pasture grass. Anoplocephala perfoliata colonises the gastrointestinal tract (especially ileocecal valve). In many cases, an infection with Anoplocephala perfoliata is asymptomatic. In severe cases, signs such as colic, diarrhoea and weight loss can occur.

Detection: - combined flotation and sedimentation method. As the eggs are excreted very irregularly, a negative faecal result cannot rule out an infection. Antibody detection is more sensitive.
- antibody detection by ELISA from serum for A. perfoliata

Results: microscopy: day of sample receipt (Mon – Fri)
ELISA: 5 working days after sample receipt

4.4.5 Oxyuris equi (Awl Tail)

Infections mainly occur in adult and older horses. Affected horses are characterised by egg cords and bark in the anal area as well as itching and tail rubbing.

Detection: anal impression smear

Results: day of sample receipt (Mon – Fri)

4.4.6 Strongyloides westeri (Dwarf Threadworm)

The clinical significance is controversial, patent infections occur predominantly in foals up to 6 months of age. The infections are often latent, with unfavourable courses resulting in diarrhoea, weakness and runting.

Detection: - flotation method (only useful from fresh faecal samples)
- larval culture

Results: flotation: day of sample receipt (Mon – Fri)
larval culture: 10 – 12 calendar days after sample receipt

4.4.7 Protozoa: Giardia, Eimeria leuckarti, Cryptosporidium parvum

Protozoa generally only lead to clinical signs in foals and young animals.

Giardia

Detection: - microscopic detection using the enrichment method is less sensitive than PCR and ELISA, but serves to detect a pathogenic infection
- PCR
- ELISA

Results: microscopy, ELISA: day of sample receipt (Mon – Fri)
PCR: 1 – 3 working days after sample receipt

Caution:
After therapy, false positive results are possible with PCR or ELISA detection. A follow-up test should therefore be carried out after 3 weeks at the earliest.

Eimeria leuckarti (foals, especially 2nd half of the suckling period, weanlings)

Detection: microscopically after flotation/enrichment

Results: day of sample receipt (Mon – Fri)

Cryptosporidium parvum (suckling foal, especially 2nd – 4th week of life, < 6 months)

Infected foals show diarrhoea with malabsorption and exsiccosis. Co-infections with other pathogens such as rotaviruses etc. lead to a worsening of the course of the disease.

Detection:
- coproantigen EIA
- PCR

Results: 1 – 3 working days after sample receipt

4.4.8 Fasciola hepatica (Liver Fluke)

The liver fluke parasitises in the bile ducts of various mammals. The eggs are excreted with the faeces and undergo a development cycle in which the dwarf mudsnail serves as an intermediate host. The metacercariae are then ingested by the final host. There they penetrate the intestinal wall, migrate through the liver and reach the bile ducts, where they develop into adult liver flukes. In horses, infestation is rare; risk factors include grazing on damp pastures or access to natural water, especially in mixed grazing with sheep or cattle. The infection is often asymptomatic. The following signs are associated with a liver fluke infestation: diarrhoea, apathy, anorexia, emaciation, poor performance. Increased liver enzyme activity is possible.

Detection:
- sedimentation method from faeces. Problem: in horses, egg excretion may remain low or absent.
- antibody detection via ELISA from serum

Results: sedimentation: day of sample receipt (Mon – Fri)
antibody detection: 5 – 7 working days after sample receipt

4.4.9 Dictyocaulus arnfieldi (Lungworm)

Infection requires grazing with donkeys or mules that excrete lungworms. Coughing is the predominant sign.

Detection:
- migration technique according to Baermann from the faeces
  However: in horses, the development of adult stages is rare (foals, yearlings), which is why there is usually no egg excretion. This detection method is well suited for donkeys.
- detection of larvae from bronchoalveolar lavage (BAL)

Results: 1 – 2 working days after sample receipt

4.5 PCR Profiles

For all PCR profiles for horses, the results are transmitted 1 – 3 working days after receipt of the sample.

4.5.1 Abortion

PCR detection: equine herpesvirus 1 and 4 (EHV1, EHV4), equine arteritis virus (EAV), leptospira, Listeria monocytogenes

Material: abortion material, swab without medium (genital tract)

4.5.2 Anaemia small

PCR detection: Anaplasma phagocytophilum, babesia (piroplasms incl. species differentiation)

Material: EB

4.5.3 CEM

Information regarding the CEM profiles (mare and stallion) can be found in Chapter 5.6, page 55.

4.5.4 Diarrhoea Pathogens (foal)

PCR detection: rotavirus A, Clostridium perfringens netF (gene), Clostridioides difficile toxin A+B (gene), Rhodococcus hoagii (formerly R. equi) incl. vapA, equine coronavirus, Lawsonia intracellularis

Material: faeces

4.5.5 Eyes

- **Eye**
  PCR detection: equine herpesvirus 2 and 5 (EHV2, EHV5)
  Material: swab without medium (conjunctiva)
- **Uveitis**
  Antibodies: leptospira
  PCR detection: leptospira, equine herpesvirus 1 (EHV1)
  Material: aqueous humour 1 ml

4.5.6 Hepatotropic viruses

PCR detection: equine parvovirus, equine hepacivirus

Material: EDTA blood, serum or liver tissue (also embedded in paraffin)

4.5.7 Neurology

Information regarding the profile Neurology can be found in Capter 2.20, page 27.

4.5.8 Respiratory

- Respiratory Foal
  - PCR detection: equine herpesvirus 1 and 4 (EHV1, EHV4), Influenza A virus and Rhodococcus hoagii (formerly: R. equi)
  - Material: nasal swab (deep) without medium or tracheobronchial secretion (TBS)/bronchoalveolar lavage (BAL)

- Respiratory I
  - PCR detection: equine herpesvirus 1 and 4 (EHV1, EHV4), Influenza A virus, Streptococcus equi equi/zooepidemicus
  - Material: nasal swab (deep) without medium or tracheobronchial secretion (TBS)/bronchoalveolar lavage (BAL)

- Respiratory II
  - PCR detection: equine herpesvirus 1 and 4 (EHV1, EHV4), Influenza A virus, Streptococcus equi equi, equine coronavirus
  - Material: nasal swab (deep) without medium or tracheobronchial secretion (TBS)/bronchoalveolar lavage (BAL) and faeces

- Respiratory III
  - PCR detection: equine herpesvirus 1 and 4 (EHV1, EHV4), Streptococcus equi equi/zooepidemicus
  - Material: nasal swab (deep) without medium or tracheobronchial secretion (TBS)/bronchoalveolar lavage (BAL)

- Respiratory IV
  - PCR detection: equine herpesvirus 1 and 4 (EHV1, EHV4)
  - Material: nasal swab (deep) without medium or tracheobronchial secretion (TBS)/bronchoalveolar lavage (BAL), EDTA blood (fever)

4.5.9 Skin

PCR detection: dermatophytes, Dermatophilus congolensis

Material: scabs; skin scrapings or skin biopsies

5. Export-relevant Infectious Diseases

This chapter will briefly discuss the diseases which are required for so-called "export-tests". Requirements vary from country to country. The embassies or consulates of the respective import countries should have information available regarding the current regulations. Mostly, these are also available fully up-to-date on the websites of these countries.

It is essential to take into account the specified test duration, which may be several days, when collecting and sending samples.

You can download our special submission form for export tests ('Equine Reproduction and Trade') in 'MyLab' or on request.

5.1 African Horse Sickness Virus (AHSV)

As the name suggests, AHS is an endemic viral disease in equids particularly in Central Africa; sporadic outbreaks have been observed in the Middle and the Near East as well as in Southern Europe. Generally, the disease is transmitted by Culicoides spp., but also by Culex, Anopheles, Aedes and ticks. All secretions, intestines and the blood of infected animals are infectious.
A distinction is made between a subclinical, febrile form, a subacute cardiac form, an acute pulmonary form and a mixed form; CNS manifestation is rare.
All organ manifestations are accompanied by oedema and haemorrhages. The mortality rate is about 70 – 95 % in horses, approx. 50 % in mules and approx. 10 % in donkeys.

Material: antibody detection in serum by cELISA

Results: 5 working days after sample receipt

Note:
AHS is an epizootic disease that is notifiable upon suspicion in most countries!

5.2 Burkholderia mallei (Glanders)

Glanders is an equine disease caused by Burkholderia mallei, which also has zoonotic potential: Apart from humans, wildcats (zoos!), camelids, bears, wolves and dogs are susceptible. Cattle, sheep and pigs are resistant. The disease is either acute (especially in donkeys and mules) with high fever and respiratory symptoms and death after a few days. In horses, glanders mostly takes a chronic course with nodules and ulcerations on the skin, the mucous membrane and the inner organs. Chronically and subclinically infected animals are a dangerous source of infection. All secretions of the respiratory tract and the skin are infectious; the incubation period ranges from a few days to many months.
In Europe, glanders is considered eradicated; but it does occur in different Asian, African and South American countries.

Material: antibody detection in serum by CFT

Results: 5 working days after sample receipt

Note:
Glanders is an epizootic disease that is notifiable upon suspicion in many countries!

5.3 Equine Arteritis Virus (EAV) (Equine Viral Arteritis, EVA)

For further information, see Chapter 4.2.2, page 39.

Material:
- antibody detection in serum by VNT
- RNA detection in semen by PCR

Note:
In Germany, EVA is a reportable disease (horses, donkeys, etc.).

5.4 Equine Infectious Anaemia Virus (EIAV)

For further information, see Chapter 4.2.6, page 42.

Material: antibody detection in serum by Coggins test (= agar gel immunodiffusion test)

Note:
EIA is an epizootic disease that is notifiable upon suspicion in most countries!

5.5 Salmonella Abortusequi

Transmission occurs orally, rarely through mating. With regard to miscarriages, this pathogen does not currently play a role in Germany anymore.

Material: antibody detection in serum by MAT

Results: 5 working days after sample receipt

Note:
Authorities must be notified if Salmonella Abortusequi is detected.

5.6 Taylorella equigenitalis (CEM = Contagious Equine Metritis)

CEM is caused by Taylorella equigenitalis. Transmission particularly occurs during mating but also indirectly by contaminated instruments or other items. Stallions may latently carry the pathogen on the mucosa of the penis without becoming clinically ill. In mares, an infection usually leads to endometritis/cervicitis with mucopurulent vaginal discharge and to reduced fertility.
The sampling sites result from the predilection sites of the pathogen:
- In stallions: penile sheath, urethra and fossa glandis
- In mares: fossa clitoridis and med. & lat. sinus clitoridis.

Transportation of these samples in medium with charcoal (e.g. Amies) is mandatory. Current animal health regulations require an examination by bacterial culture for at least 7 days (Canada: 14 days) due to the very slow growth of the microaerophilic bacterium. The CEM culture can also be selected as a profile in combination with the Reproductive Health test. To get a faster result, a PCR can be performed for Taylorella equigenitalis.

Within the EU, the detection of Taylorella equigenitalis by PCR is also recognised as a suitable test method in addition to the bacteriological examination.
In accordance with Council Directive 92/65/EEC, we offer a CEM PCR profile for 3 samples (stallion) as part of export tests within the EU. This profile includes 3 single tests by PCR for Taylorella equigenitalis from the 3 sites that are specified. There is a corresponding CEM PCR profile available for mares (2 sites).
For CEM export tests, the submission of 2 swabs (mare) or 3 swabs (stallion) with charcoal medium is also required for PCR.

In addition, we also offer 2 expanded profiles for both stallion and mare. These expanded profiles include the examination of sperm or a sample from the prepuce for stallions and of a cervix swab for mares in addition to the examination of the above-mentioned swabs of 3 (stallion)/2 (mare) sites.

Results: culture: 1 week after sample receipt (Canada 2 weeks)
PCR: single assay 1 – 3 working days (Mon – Fri) after sample receipt
CEM Profiles 1 – 2 working days (Mon – Sat) after sample receipt

In Germany, there is an obligation to inform the authorities, if Taylorella equigenitalis is detected.

5.7 Theileria equi/Babesia caballi (Piroplasmosis, Babesiosis)

For further information, see Chapter 4.3.2, page 47

Material: antibody detection in serum
by CFT (mandatory for most countries)
or cELISA (for export to the USA)
or IFAT (only available for export tests)

5.8 Trypanosoma equiperdum (Dourine)

Dourine is a chronic or acute infectious disease in equines, which is transmitted directly between animals during mating. Infected equines are the only natural reservoir; the pathogens are present in the genital secretions of both mares and stallions. Incubation period, severity and duration of the disease vary considerably. Subclinical infections are possible; donkeys and mules are more resistant to the pathogen. Clinically, affected animals show inflammation of the outer genitals with depigmentation of the mucosa up to peripheral-neurological disorders/paralysis.
Particularly in Asia and Africa, Trypanosoma is still widespread; Central Europe is currently considered free from Trypanosoma equiperdum.

Material: antibody detection in serum by CFT

Results: 5 working days after sample receipt

Note:
Dourine is an epizootic disease that is notifiable upon suspicion in many countries!

6. Microbiology and Parasitology

6.1 Bacteriological Examination

Material: swab with transport medium

Please be sure to indicate the localisation of the sampling so that we can add selective media if necessary and evaluate the antibiograms according to CLSI. In the case of aerobic bacteriology, the antibiograms are prepared automatically if the germs are clinically relevant, unless they are actively cancelled (number 2202 is available for print submission forms and number 220200 for online submission forms). In countries where an antibiogram is mandatory, there is no option to deselect the antibiogram. Especially in cases of abscesses or wound infections, testing for anaerobes should also be done. In purulent conditions, it is important to collect the sample from the area of transition between healthy and infected tissue. You can select the anaerobic culture individually or together with the aerobic bacteriology as a more cost-effective combination service (1061).

The antibiograms for the anaerobic germs must be actively ordered within 48 hours (service 727). In countries where an antibiogram is mandatory, the antibiogram of anaerobes is also performed automatically.

Results: aerobic culture: 2 – 3 working days after sample receipt
anaerobic culture: 4 – 7 working days after sample receipt

6.2 Mycological Examination

Material: swab with transport medium

Yeasts and moulds are detected. The examination takes up to one week. An antimycogram of yeasts can be prepared on request. In some countries the antimycogram is also performed automatically for yeasts.

6.3 Skin Diseases

6.3.1 Bacteriological Examination of the Skin

Material: swab with transport medium (see Chapter 6.1, page 57).

6.3.2 Dermatophilus congolensis

Dermatophilus congolensis is the causative agent of "mud fever". The disease develops when pre-injured skin (micro traumata!) is permanently exposed to wet conditions. Signs are scurfs, thick scabs and alopecia. Detection is done by PCR from skin, crusts, tissue. The test can be requested individually or together with the PCR for dermatophytes as Skin Profile.

Material: scabs

Results: 2 – 4 working days after sample receipt

6.3.3 Mycological Examination of the Skin

For mycological examinations, sampling of hairs from the area of transition between intact and altered skin is recommended in addition to skin scrapings. It is best to send in skin scrapings and hairs in sterile tubes or small paper bags.
In addition to the fungal culture on a special growth medium, a slide for microscopic examination is prepared. An antimycogram can be performed on special request if yeasts are detected.
For the diagnosis of dermatophytosis, a routine **dermatophyte PCR** test is available in addition to fungal culture. This molecular method helps to significantly reduce the time it takes to make a diagnosis, allowing for an early antimycotic therapy. The dermatophyte PCR is approximately as sensitive as fungal culture and it is not susceptible to moulds, but negative results cannot completely rule out an infection – neither in culture nor in PCR.

Material: cultural examination: scrapings, hairs, crusts, swabs with transport medium
PCR: scrapings, hair, crusts

Results: culture: up to 3 weeks after sample receipt
PCR: 2 – 4 working days after sample receipt

6.3.4 Parasitological Examination of the Skin

The parasitological examination is carried out microscopically and serves to detect ectoparasites such as mites, lice or hair lice.

Material: scrapings, plucked hairs, tape swab

If mites are suspected to be the causative agent, the depth of the scraping has to be adapted to the living habits of the mites:
Chorioptes bovis (leg and tail mange): superficial skin scraping
Psoroptes equi (head mange): combed out hairs, ear detritus
Demodex caballi (eye lids, mouth)/Demodex equi (whole body): deep skin scraping

Results: day of sample receipt (Mon – Fri)

6.4 Pre-breeding Examinations

6.4.1 Reproductive Health

In addition to gynaecological/andrological examinations of all animals that are intended for breeding, the microbiological examination of swabs is used to detect those animals whose genitalia are colonised by pathogenic and facultative pathogenic agents.
As these infections usually progress subclinically, only bacteriological/mycological examination can provide insight into the microorganisms involved, allowing for a specific treatment to make the horses fit for breeding.
Please note: if there are any clinical symptoms please choose the bacteriological examination. Otherwise we will only perform an antibiogram if the following bacteria are detected (as these are considered pathogenic, regardless of symptoms):

- Actinobacillus equuli
- Bordetella bronchiseptica
- E. coli var. haemolytica
- Klebsiella
- Pseudomonas aeruginosa
- Raoultella ornithinolytica
- Rhodococcus hoagii (formerly: R. equi)
- Staphylococcus aureus
- β-haemolytic Streptococci

Clinically healthy mares in which a high level of
- E. coli in pure culture
- Serratia marcescens in pure culture
- yeasts
- moulds

is detected should also be treated – for hygienic reasons.

A "certificate of good health" can only be issued by the veterinarian who examined the animal taking into account the clinical as well as the bacteriological findings.
We offer the test for reproductive health as a single examination, in combination with CEM testing (detection of Taylorella equigenitalis by culture) or combined with a mycological examination or with a mycological examination and an endometrial biopsy (see Chapter 11.1.1, page 85).

Material: mare: Swab with medium (cervix or uterus)
stallion: Swab with medium (penile sheath, urethra, glans penis)

If CEM is to be tested, too, a swab with charcoal transport medium is required (e.g. Amies). If CEM should be detected by PCR as well as by bacterial culture, a separate swab is required for each examination method.

Results: 2 – 3 working days after sample receipt (culture).

6.4.2 Taylorella equigenitalis/CEM

See Chapter 5 "Export-relevant Infectious Diseases", page 55.

6.4.3 Taylorella asinigenitalis

Taylorella (T.) asinigenitalis, a non-motile gram-negative rod-shaped bacterium, is closely related to T. equigenitalis and is found on the genital mucosa of solipeds (mainly donkeys, less frequently horses). The pathogen is detected more frequently in male animals than in mares. Stallions act as asymptomatic carriers. Transmission occurs via mating. T. asinigenitalis is usually described as apathogenic, but there are also pathogenic strains that can cause severe purulent endometritis in mares. In donkey mares, the infection is usually asymptomatic. For a comprehensive breeding hygiene examination, the additional PCR test for T. asinigenitalis – in addition to the PCR for T. equigenitalis – is recommended in order to recognise future outbreaks at an early stage.

Detection: PCR from swabs taken from the genital mucosa (swab without or with medium), semen

Results: 1 – 3 working days after sample receipt

6.5 Faecal Analysis

6.5.1 Microbiological Faecal Analysis

The bacteriological faecal analysis detects facultative pathogenic organisms as well as salmonella. It is included in some of our profiles: For horses, the small and the large Faecal Profile are available, and for foals, the Foal Faecal Profile. We also offer the Foal Diarrhoea Pathogens PCR profile, which includes testing for rotavirus A, Clostridium perfringens netF, Clostridioides difficile toxin A+B, Rhodococcus hoagii (R.equi) incl. vapA, equine coronavirus and Lawsonia intracellularis.

Material for the profiles: faeces

6.5.2 Maldigestion/Malabsorption

To find out whether or not food components are sufficiently broken down in the gastrointestinal tract, the particle size can be determined.

Results: day of sample receipt (Mon – Fri)

6.5.3 Parasitological Faecal Analysis

The parasitological examination of faecal samples from horses includes flotation and enrichment by SAFC (sodium acetate-acetic acid-formalin concentration). The number of detected parasite stages is indicated as "low, medium and high-grade". In order to counteract the increasing development of resistance of endoparasites against anthelmintics, alternative deworming strategies have been devised. If the concept of selective deworming is used in a horse population, the faecal sample should be examined by using the modified McMaster method. Here, the exact egg count/amount of oocysts per gram of faeces is determined (epg/opg) by counting in a counting chamber. Deworming is only carried out if there are > 200 strongyle eggs per gram of faeces in a horse.
However, in case of tapeworm infestation (Anoplocephala perfoliata) or if nematodes (Parascaris equorum) are detected, deworming should generally be performed, regardless of the degree of infestation.

Equine Endoparasite Profile, including flotation, SAFC and modified McMaster.

As worm eggs are shed intermittently, it is recommended to send in a 3-day pooled faecal sample in order to increase the probability of detection.

Note:
It is not possible to differentiate between large and small strongyles based on the eggs. Instead, a larval culture would be required. Larval culture should always be carried out when large strongyles are suspected and at least once a year in herds that use the selective deworming method.
Information on specific endoparasitoses can be found in Chapter 4.4, page 47.

6.5.4 Dysbiosis Analysis

The aim of dysbiosis analysis is to recognise dysbiotic conditions. For this purpose, quantitative PCR is used to analyse selected marker germ groups that provide information about the composition of the intestinal microbiota.

Results: 3 – 5 working days after sample receipt

6.6 Autovaccines

Following a bacterial culture and depending on the clinical issue, there is basically the possibility to produce an autovaccine (so-called farm-specific vaccine according to the German Epizootic Diseases Act). For this, an individually adjusted bacterial lysate is produced which can be applied orally or subcutaneously or by inhalation – depending on the affected organ system and the clinical signs. The immune system is stimulated both specifically and non-specifically.
In horses with "faecal water syndrome", for example, an orally applied autovaccine can induce the production of secretory IgA, thus stabilising the mucosal barrier.
Autovaccines can be ordered within one week after the arrival of the samples, as pathogens from the bacterial culture are stored for 7 days.
A veterinary **prescription** is required to order an autovaccine; billing the animal owner is therefore not possible. The delivery time to the veterinary pharmacy is approximately 3 weeks.

7. Urinalysis

Urinalysis in addition to blood/serum analysis is used for laboratory diagnostic workup of urinary tract diseases. But urinalysis is also useful regarding other issues: e.g. the determination of glucose in the urine of hyperglycaemic horses or the fractional electrolyte excretion, which also can be interpreted with regard to the electrolyte supply of the horse in case of muscular problems.
Please note: information concerning SDMA, a helpful parameter for the early detection of renal dysfunction, can be found in Chapter 3.4, page 29.

7.1 Urinalysis including Urinary Sediment

Specific gravity, protein, haemoglobin/myoglobin, pH, bilirubin, urobilinogen, glucose, nitrite, ketone bodies as well as erythrocytes, leukocytes, bacteria, yeasts, cylinders, epithelial cells, crystals

Material: urine 5 ml

Method: dry chemistry, photometry, refractometry

7.2 Protein/Creatinine Ratio

For the diagnosis of nephropathies and loss of protein

Material: urine 1 ml

Method: photometry

7.3 γ-GT/Creatinine Ratio

Shows the early stage of a tubular disease and is indicated in case of acute disease.

Material: urine 1 ml

Method: photometry

7.4 Fractional Electrolyte Excretion (FE)

The FE is used to diagnose a dysfunction of the renal tubules. In horses with healthy kidneys, the net excretion of an electrolyte in the urine is regulated by 1) the glomerular filtration rate and 2) the tubular reabsorption. If the electrolyte excretion is put into relation with the creatinine excretion (here GFR = excretion), it indicates the FE of the electrolyte.
If tubular reabsorption fails, the FE of one or more electrolytes usually increases and its FE values will be above the normal range.

Interpretation:
Persistent or repeated FE increases of one or more electrolytes (especially Na and P) indicate tubular malfunction.

Material: non-haemolysed S + urine – samples collected at the same time

Parameter: FE of Na, K, P and Cl

Method: photometry

7.5 Bacterial Culture – Urine

In case of clinical suspicion of a urinary tract infection or if urinalysis and sediment are suspicious, a bacteriological examination is indicated. The urinary culture test includes a microbial count as well as pathogen differentiation. If the results are clinically relevant, an antibiogram will automatically be performed; please also refer to the notes in Chapter 6.1, page 57).

Material: clean-catch midstream urine, catheter urine

8. Endocrinology

8.1 Sex Hormones
8.1.1 Oestradiol
8.1.2 Progesterone
8.1.3 Testosterone

8.2 Hormonal Pregnancy Diagnosis
8.2.1 PMSG/eCG
8.2.2 Luteo-placental Shift
8.2.3 Oestrone Sulphate

8.3 Diagnosis of Ovarian Tumours
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8.4 Diagnosis of Cryptorchidism
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8.5 PPID (Equine Cushing)
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8.6 Equine Metabolic Syndrome (EMS)
8.6.1 Analysis of Basal Insulin and Basal Glucose
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8.7 Hypoadrenocorticism
8.7.1 ACTH Stimulation Test

8.8 Thyroid Gland
8.8.1 TRH Stimulation Test with Determination of T4

8.1 Sex Hormones

8.1.1 Oestradiol

Produced synchronously to the oestrus cycle in the ovarian follicles ("oestrus hormone"); during pregnancy, there is massive oestrogen biosynthesis of the foeto-maternal unit.

Material: S or plasma 0.5ml

Results: day of sample receipt (Mon – Sat)

Method: CLIA

8.1.2 Progesterone

Synthesised by luteal cells of the corpora lutea (c.l.): values ≥ 1 ng/ml indicate luteal function; c.l. during the cycle and especially during pregnancy almost always show much higher values.
During an intact early pregnancy, the concentration of progesterone in the blood should not drop below 4 ng/mg.
Laboratory diagnosis cannot differentiate between cyclic corpora lutea and corpora lutea in pregnancy. If progesterone is used for pregnancy diagnosis on day 18 – 20 post ovulationem and a corpus luteum is detected, it only means that the mare will not return to oestrus at the expected time.

Material: S or HP 0.5 ml

Results: day of sample receipt (Mon – Sat)

Method: CLIA

8.1.3 Testosterone

It is produced in the testicular interstitial cells of Leydig and, to a small extend, also in the adrenal cortex. Sampling should take into account the circadian fluctuation: testosterone levels are low in the morning and highest in the afternoon.
Mares also produce small amounts of testosterone in the ovaries and adrenal cortex.

Material: S or HP 0.5 ml

Results: 1 – 3 working days after sample receipt

Method: LCMS

Note:
Interpreting the analysis of sex hormones can only be done if the results of a clinical examination are considered as well. In some cases, follow-up examinations may become necessary.

8.2 Hormonal Pregnancy Diagnosis

Sometimes, rectal palpation for pregnancy detection is impeded by very pragmatic matters: ponies/miniature horses, aggressive animals, feral and zoo animals, lesions of the rectum, etc. For such situations, we have two pregnancy-specific hormones available in the equine practice:

8.2.1 PMSG/eCG

It is produced by the "endometrial cups" approximately between day 35 and day 120 of pregnancy (in individual cases even longer) with highest values between day 60 and 75. After early embryonic loss, endometrial cups will continue to secrete PMSG for weeks and the test will yield false positive results concerning pregnancy. We therefore recommend re-testing the mares after day 110 of pregnancy by determining their oestrone sulphate levels (see below). The test is also possible with the donkey mare.

Material: S or HP 0.5ml

Results: 1 – 3 working days after sample receipt (horse)

Method: ELISA

Note:
We recommend sampling between day 45 and 100 post ovulationem.

8.2.2 Luteo-placental Shift

The determination of the DHP/progesterone quotient by measuring the 5 alpha-dihydroprogesterone (DHP) allows a statement to be made about the time of the luteo-placental shift between the 100th – 120th day of pregnancy. Absolute values (follow-up examinations) allow a statement to be made about placental function in the case of diseases during advanced pregnancy.

Material: S 1 ml

Results: 1 – 5 working days after sample receipt

Method: LCMS

8.2.3 Oestrone Sulphate

It is produced by the intact foeto-placental unit and thus indicates the presence of a live foetus. The hormone can be detected from approx. day 40 of pregnancy onwards in increasing concentrations; at this early stage, however, there is no reliable differentiation from cyclical hormone secretion possible. We recommend oestrone sulphate testing from day 110 onwards, since mares at this stage of pregnancy have much higher concentrations of oestrone sulphate. The test is possible for horse and donkey mares.

Material: S 1 ml, possibly HP, EP, U

Results: 1 – 3 working days after sample receipt

Method: LCMS

Preferably ≥ day 110 of pregnancy

Note:
Not all mares show the typical pattern of secretion. If the test results are borderline or inconclusive, we suggest re-testing after 3 – 4 weeks. A negative test result in mares which have definitely been pregnant for > 120 days can indicate damage to the foetus. In this case, a rectal palpation or an ultrasound examination is mandatory.

8.3 Diagnosis of Ovarian Tumours

Mares with cycle or behavioural abnormalities which also show abnormal rectal/sonographic ovarian findings are often suspected to have an ovarian tumour. In fact, ovarian tumours are among the most frequent neoplasms in horses; the most common diagnosis is granulosa cell tumour (GCT). This kind of tumour is able to secrete oestradiol and testosterone. The determination of these hormones can therefore also be used to make the diagnosis. It must be taken into account, though, that on the one hand, mares with such a tumour may have normal hormone levels and on the other hand, elevated oestradiol and testosterone levels may be found in certain cycle abnormalities or in pregnant mares.

8.3.1 Anti-Müllerian Hormone (AMH)

AMH is a glycoprotein which is crucial for sexual differentiation during embryonic development. In female animals, AMH is secreted by the granulosa cells of preantral and small antral follicles. Since granulosa cell tumours (GCT) are the most frequent tumours of the genital tract in mares, it made sense – as in human medicine – to pursue this diagnostic approach in mares as well. Mares with GCT showed significantly higher levels of AMH than healthy mares. Regarding GCT diagnosis, AMH has a sensitivity of 95 %.

Material: S 0.5 ml, centrifuged and pipetted soon after sampling. Cooling the sample is recommended.

Results: day of sample receipt (Mon – Sat)

Method: CLIA

Interpretation:
"Intact" mares: < 4 ng/ml – individual levels
Ovariectomised mares: < 0.1 ng/ml
Mares with GCT: > 7 ng/ml
Borderline: 4 – 7 ng/ml

No validated reference values are available for this parameter for granulosa cell tumour diagnostics in the donkey mare.

8.3.2 Granulosa Cell Tumour Profile

This profile helps you in the diagnosis of hormonal disorders or hormone-producing ovarian tumours (granulosa-theca cell tumours), which can lead to behavioural changes and unrideability in mares.
It includes the parameters anti-mullerian hormone, progesterone and testosterone.

Material: S 2 ml, cooling recommended

Results: 1 – 3 working days after sample receipt

Method: CLIA, LCMS

8.4 Diagnosis of Cryptorchidism

Detection of cryptorchid or incompletely neutered male horses. Single determinations of testosterone levels are often of little significance due to pronounced circadian and seasonal fluctuations.

8.4.1 HCG Stimulation Test/"Cox Test"

For a long time, HCG stimulation test was considered gold standard for the diagnosis of cryptorchidism.

Principle:
HCG has LH-like effects

Test procedure:
- In the morning: first blood collection = testosterone baseline value
- Directly followed by i.v. injection of 5000 – 10000 IU HCG/horse
- + 60 min: second blood collection = stimulation value

Material: S each 0.5 ml

Results: 1 – 3 working days after sample receipt

Method: LCMS (determination of testosterone level with 2 samples)

Interpretation:
Completely castrated horses show very low levels and a lack of stimulation. A significant rise in testosterone levels indicates the existence of testosterone-producing tissue. At the same time, the absolute values should also be taken into consideration.
The determination of testosterone / the HCG stimulation test can also be carried out on male donkeys.

8.4.2 GnRH Stimulation Test

The use of a releasing hormone additionally tests the hypothalamus pituitary axis. This is not really necessary for the mere diagnosis of cryptorchidism.

Test procedure:
- In the morning: first blood collection = testosterone baseline value
- Directly followed by i.v. injection of 0.04 mg GnRH/horse
- + 60 min: second blood collection = stimulation value

Material: S each 0.5 ml

Results: 1 – 3 working days after sample receipt

Method: LCMS (determination of testosterone level with 2 samples)

Interpretation:

Depending on the clinical issue

8.4.3 Anti-Müllerian Hormone (AMH)

In stallions, AMH is expressed in the Sertoli cells. Levels stay high until puberty, then decrease parallel to increasing testosterone secretion. Nevertheless, stallions and cryptorchids can clearly be differentiated from castrated animals. AMH in male animals is a useful biomarker for the presence of testicular tissue and can thus be used for the diagnosis of cryptorchidism. This test also works in young castrated animals.
The determination of AMH is also possible in male donkeys for cryptorchid diagnostics.

Material: S 0.5 ml, centrifuged soon after sampling and pipetted off without any cells. Cooling is recommended.

Results: day of sample receipt (Mon – Sat)

Method: CLIA

Interpretation:
Values < 0.1 ng/ml indicate complete castration
Values 0.1 to 2 ng/ml are to be considered borderline
Values > 2 ng/ml indicate the presence of testicular tissue

Male donkey castrated: < 14.7 +/- 2.4 ng/ml indicates complete castration; values above the reference range indicate the presence of testicular tissue in donkeys. An HCG stimulation test should be performed to confirm this.

8.5 PPID (Equine Cushing)

Equine Cushing's disease is the most frequently diagnosed endocrinopathy in horses. It is a typical disease of old horses and ponies. It is caused by pituitary adenoma – more precisely: pars intermedia hyperplasia (PPID = pituitary pars intermedia dysfunction). In horses, nearly solely pituitary Cushing's is of clinical relevance.
Clinical signs are: hirsutism/hypertrichosis, polydipsia/polyuria, weight loss with simultaneous fat redistribution, depression/lethargy, laminitis, insulin resistance, immunosuppression and hyperhidrosis (pathologically increased sweating).
Below, you will find our endocrinological tests for the diagnosis of PPID. Further information on the **PPID Profile**, which includes the evaluation of hormones as well as clinical chemical parameters, can be found in Chapter 2.11, page 24.

8.5.1 ACTH Analysis

In horses, the test has a high diagnostic accuracy and is considered the best diagnostic alternative to the TRH Stimulation Test. The determination of endogenous ACTH is especially indicated if the horse is domiciled far away or in laminitic horses.

Test procedure:
Stress-free blood collection: EDTA plasma is required, which is centrifuged promptly and pipetted off without any cells! Cooling is recommended, the temperature of the samples must not rise above room temperature. The sample should arrive at the laboratory the next day.

Material: EP 0.5 ml (cooled)

Results: day of sample receipt (Mon -Sat)

Method: CLIA

Interpretation of basal ACTH:
Due to seasonal differences, the limit values (in pg/ml) must be interpreted as follows based on the recommendations of the Equine Endocrinology Group 2023:

Season	PPID negative	PPID questionable	PPID positive
December – June	< 15	15 – 40	> 40
July	< 15	15 – 50	> 50
August	< 20	20 – 75	> 75
September – October	< 30	30 – 90	> 90
November	< 15	15 – 40	> 40

The values should be seen as a guide; individual variability may cause discrepancy. ACTH values should always be assessed taking into account the clinical signs and the breed. Questionable or negative results in the case of existing clinical suspicion should

be confirmed by means of a TRH Stimulation Test in the months of January – June.
Reference values for donkeys, see Chapter 14.3, page 128.
It is important for correct results to adhere to the key pre-analytical data: prompt centrifugation and pipetting of the EDTA plasma and cooling of the samples (see above)!

Note:
Endogenous ACTH should not be tested during acute laminitis! Testing of clinically healthy horses is not recommended.

8.5.2 TRH Stimulation Test with ACTH Analysis

Test with high specificity and sensitivity for for PPID diagnostics, recommended by the Equine Endocrinology Group. It is indicated if the results of the ACTH Analysis or the Suppression Test do not correlate with clinical findings or are inconclusive.

Test procedure:
- First blood collection = baseline value
- Directly followed by slow i.v. injection of 1 mg TRH for horses >250 kg (horses <250 kg: 0.5 mg)
- Second blood collection after exactly 10 min = stimulation value
- Third blood sample can additionally be taken 30 minutes after TRH administration

Material: EP 0.5 ml (cooled) – see pre-analytics in Chapter 8.5.1, page 71

Results: day of sample receipt (Mon -Sat)

Method: CLIA

Interpretation:
For basal values, see Chapter 8.5.1, page 71.
Cut off 10 min after stimulation: <100 pg/ml; borderline: 100-200 pg/ml;
positive: >200 pg/ml
Cut off 30 min after stimulation: <40 pg/ml; borderline: 40-90 pg/ml;
positive: >90 pg/ml
These values apply to the months of January to June.
The stimulation test is not recommended from July to December. If the result is negative during this period (<100 pg/ml after 10 min, <40 pg/ml after 30 min), the diagnosis of PPID is very unlikely.

And a note on cortisol determination in the context of PPID diagnostics:
In most horses suffering from PPID, cortisol levels are within reference range, as in affected animals, mainly the circadian rhythm of secretion is disturbed. Healthy horses show highest cortisol concentrations in the early morning, lowest in the afternoon/early evening. Further factors influencing endogenous cortisol are stress, exercise, pain, etc. With regard to PPID, determination of cortisol is only useful if evaluated in connection with a function test (see Chapter 8.5.3, page 73)!

8.5.3 Overnight Dexamethasone Suppression Test (ODST)

This test can be performed as an alternative to the TRH Stimulation Test.

Principle:
Exogenous dexamethasone suppresses endogenous corticoid secretion by negative feedback on the pituitary gland – in healthy horses. In PPID horses, there is no feedback as the mutated pars intermedia cells have no corticoid receptors.

Test procedure:
- Blood collection between 4 pm and 6 pm = cortisol baseline value
- Directly followed by i.m. injection of 2 mg/50kg bdw of dexamethasone
- (Perhaps additional blood collection after 15 hours. However, long-term Suppression after 18 – 20 hours is decisive.)
- Blood collection between 10 am and 1 pm the next day = suppression value

Material: S each 0.5 ml

Results: day of sample receipt (Mon – Sat)

Method: CLIA (in both samples)

Interpretation:
In healthy horses, values are suppressed to well below 10 ng/ml.

Note:
In late summer/autumn, even healthy horses may suppress inadequately!

8.6 Equine Metabolic Syndrome (EMS)

EMS is a severe disturbance of the carbohydrate and lipid metabolism resulting in insulin dysregulation (ID): Increased insulin secretion (partly) compensates for decreased insulin efficiency.
EMS comprises adipositas, insulin dysregulation and laminitis (either anamnestic or present).

Clinical presentation:
- Mainly middle-aged or old horses (approx. 5 – 15 years) are affected, especially easy-keeper breeds such as Ponies, Arabian Horses, Fjord Horses, Mustangs, "Baroque Horses", etc.
- Laminitis: subtle to acute
- Adipositas (approx. 10 % of the affected horses have a "lean" appearance, though)
- Polydipsia/polyuria
- Reduced fertility
- Recurrent colic

The diagnosis is based on the detection of ID; in older horses, PPID may need to be excluded or clarified.
Information on the **EMS Profile**, which includes the evaluation of insulin as well as other clinical chemical parameters, can be found in Chapter 2.12 , page 24.

8.6.1 Analysis of Basal Insulin and Basal Glucose

Material: S 1 ml, cooled (insulin) resp. NaFB 1 ml (glucose)
Both samples should be collected from the fasted animal (hay/straw only ration)!

Results: day of sample receipt (Mon – Sat)

Method: CLIA, photometry

Interpretation:
Reference range for basal insulin:
< 15 µU/ml: negative for ID
15 – 35 µU/ml: suspicious for an ID with existing clinical signs
> 35 µU/ml: positive for an ID

In donkeys, an insulin value of up to 14 µU/ml is considered physiological.

Note:
To determine the insulin level, non-haemolysed serum is required (centrifuged promptly after blood collection and pipetted off without any cells), as otherwise leukocytic proteases will lead to decreased values. The sample should arrive cooled at the laboratory the day after sample collection.
Sampling should be delayed if the horse is affected by an acute, intensely painful attack of laminitis.

8.6.2 Oral Glucose Test with Insulin Determination

Test procedure:
- The horse must fast overnight (hay/straw only ration).
- In the morning, it gets: 1g/kg bdw of glucose

Blood collection to determine insulin levels after 2 hours

Material: see Chapter 8.6.1

Results: day of sample receipt (Mon – Sat)

Method: CLIA

Interpretation:
> 69 µU/ml insulin is indicative of insulin dysregulation (ID)

8.6.3 Oral Sugar Test ("Karo Light Syrup®") with Insulin Determination

Test procedure:
- Hay/straw only ration
- Administer 0.45 ml/kg bdw of Karo Light Corn Syrup® per os
- Blood collection after 60 and/or 90 minutes, measurement of insulin and glucose

Material: S each 1 ml (cooled)

Results: day of sample receipt (Mon – Sat)

Method: CLIA

Interpretation:
> 30 µU/ml insulin is indicative of insulin dysregulation

8.6.4 Insulin Tolerance Test with Glucose Determination

Test procedure:
- Fed (pasture or hay) state! Do not fast!
- Blood collection at time 0; measurement of glucose and administration of 0.10 IU/kg bdw of regular (soluble) insulin
- Blood collection after 30 minutes, measurement of glucose
- Feed immediately after last sample

Material: NaFB each 1 ml

Results: day of sample receipt (Mon – Sat)

Method: photometry

Interpretation:
Healthy horses show blood glucose values of < 50 % of the initial value 30 min after the insulin injection and should have risen to the initial value after 2 h at the latest.

Caution:
Danger of hypoglycaemia in insulin-sensitive horses!

8.7 Hypoadrenocorticism

In horses, hypoadrenocorticism is a rare endocrine disorder. If it occurs spontaneously in horses, it is mostly primary chronic hypoadrenocorticism (Addison's disease). However, by far the most common form in veterinary medicine is secondary, iatrogenic hypoadrenocorticism caused by long-term application of exogenous glucocorticoids.

8.7.1 ACTH Stimulation Test

Evaluation of adrenal function

Test procedure:
- First blood collection in the morning = cortisol baseline value
- Directly followed by i.v. injection of 100 IU ACTH
- Second blood collection after 2 hours = stimulation value

Material: S each 0.5 ml

Results: day of sample receipt (Mon – Sat)

Method: CLIA

Interpretation:
In healthy animals, the cortisol level increases by approximately 80 %; horses with hypoadrenocorticism show very low baseline values which do not or only slightly increase after stimulation.

8.8 Thyroid gland

In horses, the thyroid gland only plays a secondary role. If at all, hypothyroidism occurs. However, hypothyroidism is often secondary, e.g. in horses suffering from PPID.
Foals are extremely sensitive to fluctuations in thyroid hormones – already intrauterine. There are also reference values for donkeys. In younger donkeys (<5 years), elevated thyroid values can sometimes be measured, in older donkeys (>11 years) lower values.

8.8.1 TRH Stimulation Test with Determination of T4

The definite diagnosis "hypothyroidism" should only be made after performing a TRH stimulation test.

Test procedure
- First blood collection = baseline value of T4
- Slow i.v. injection of 1 mg of TRH/horse or 0.5 mg of TRH/pony
- Second sample after 4 hours = stimulation value

Parameter: T4

Material: S

Results: day of sample receipt (Mon – Sat)

Method: CLIA

Interpretation:
Euthyroid horses have a 2- to 3-fold increase in T4 after 4 hours.

9. Allergy

Allergies are an increasing problem in the equine practice, especially at the beginning of the warm summer months. Horses are not only sensitised to pollen and insects – as often suspected – but also to perennial allergens like dust mites, storage mites or moulds which can be found ubiquitously in horse stables, horse feed or in the surroundings of the barn. Allergy is a threshold phenomenon, that is why clinical signs mainly occur in the warmer time of year: It takes a certain concentration of allergen(s) to induce clinical symptoms in patients. If seasonal and perennial allergens appear at the same time, they add up, thus exceeding the individual threshold level.
Horses suffering from allergy show the following clinical signs: severe pruritus, skin lesions, urticaria or respiratory diseases (equine asthma, former RAO complex "recurrent airway obstruction", and „IAD" = „inflammatory airway disease").

General information on allergy testing
The diagnosis of an allergy is a clinical diagnosis which consists of a thorough anamnesis and clinical examination. Subsequent allergy tests are only used to identify the underlying allergens in order to be able to avoid them or to perform an allergen-specific immunotherapy (hyposensitisation).
The administration of glucocorticoids or the wrong time of blood collection can considerably affect the results of the allergy test or falsify the test results. Withdrawal times of up to 3 months are recommended for injectable cortisone preparations and 6 to 8 weeks for prednisolone tablets. But also the use of locally applied cortisone ointments, creams, sprays, etc. should be stopped 2 to 4 weeks before the allergy test.
The right time to perform an allergy test is when the clinical signs have been observed for approximately 4 – 6 weeks. After this time, it can be assumed that the allergen-specific IgE antibodies are already detectable.
Furthermore, the test should be performed within or at the end of the season, as the test may yield false negative test results outside the season.

9.1 Allergy Profiles

Allergy Profile Respiratory
Determination of seasonal and perennial allergens (see Chapter 9.3).

Allergy Profile Skin
Determination of seasonal and perennial allergens, insects (see Chapter 9.3), Food Panel see Chapter 9.4).

Material (both profiles): S

Method: ELISA

9.2 Allergy Screening Test

Detects sensitisation against the 4 main allergen groups:
pollen (grasses, herbs, trees)
mites (house dust and storage mites)
fungal spores
insects

Material: S

Method: ELISA

Those groups that are positive in the screening test can then be differentiated into single allergens in the main test. All samples sent in are stored by us for 14 days. Hence, within this time frame, all necessary further tests can be ordered from a sample sent in for a screening test.

9.3 Main Tests Allergy

Seasonal Panel (pollen): Grasses and Herbs
- 6-grass mix (orchard grass, perennial ryegrass, Timothy grass, meadow fescue, Kentucky bluegrass, meadow soft grass)
- Rye (Secale cereale), mugwort (Artemisia vulgaris), lamb's quarters (Chenopodium album), English plantain (Plantago lanceolata), nettle (Urtica dioica), sorrel (Rumex rumex spp.), dandelion (Taraxacum officinalis), rape (Brassica napus), ragweed (Ambrosia elatior)

Trees and Bushes
- Hazel (Corylus avellana), alder (Alnus), poplar (Populus), birch (Betula spp.), beech (Fagus sylvatica), willow (Salix spp.)

Perennial Panel (mites & moulds): Moulds
- Alternaria alternata, Aspergillus fumigatus, Aspergillus niger, Cladosporium sp., Epicoccus nigrum, Helminthosporium sativum, Penicillium notatum, Fusarium, Ustilago, Rhizopus

Dust Mites
- Dermatophagoides farinae, Dermatoph. pteronyssinus

Storage Mites
- Acarus siro, Tyrophagus putrescentiae, Glycophagus domesticus, Lepidoglyphus destructor

Insects:
- Simulium (blackfly)
- Culex tarsalis (mosquito)
- Tabanus (horse fly)
- Musca domestica (housefly)
- Culicoides (gnat)

PAX complete:
- Food and/or Environmental
  The Pet Allergy Xplorer (PAX) test tests over 200 allergen extracts and molecular components (incl. CCD blocking) for food and environmental allergens.
  The test is offered for
  - Environmental allergens
  - Food allergens
  - Environmental and food allergens

Material: S

Method: ELISA

9.4 Further Main Tests Allergy

Feathers/Hairs/Epithelia:
Detection of IgE antibodies against epithelia of cat, dog, rabbit, guinea pig, parrot, feather mix

Food Allergy Test:
Determination of antibody titres (IgE & IgG) against 8 individual food allergens: soya, molasses, oats, maize, barley, wheat, barn and lucerne.
The definite diagnosis of a food allergy can only be made after allergen avoidance or feeding an elimination diet (only food components which have yielded a negative result for both antibodies = reaction class 0), followed by allergen provocation.

Material (both tests): S

Method: ELISA

PAX complete Food
see Chapter 9.3

9.5 Allergen-specific Immunotherapy (ASIT, hyposensitisation)

ASIT is considered the most promising therapeutic approach for allergic patients. The pathomechanism consists, among others, in the modulation of the Th2 to a Th1 cell response. Thus, it intervenes causally in the pathophysiological mechanisms of the disease.
The allergen extracts are composed individually for each patient according to the allergy test results. Our LABOKLIN allergy team will be happy to help you select the causative allergens. The allergen extracts are administered subcutaneously once a week with an initial weekly increase in dosage until the maintenance level is reached. Afterwards, the treatment intervals can be individually adjusted (up to monthly injections).
Whether ASIT has been successful should be evaluated after completing the initial and the follow-up treatment. If there is a good response, therapy should be continued for life.
In case of seasonal allergies, we recommend starting treatment at the end of the season. Individual adaptations of the dosage plan may be necessary. We will gladly advise you in such cases.

Finally, some basic comments on ASIT:
The outcome of this therapy is better the earlier it is started within the course of the disease (within 1 – 2 years after the occurrence of the first clinical signs). Middle aged horses with a short duration of clinical signs (both insect bite hypersensitivity and equine asthma) showed best therapy success; but also elder horses (> 20 years of age) still showed satisfying responses to ASIT. Very young horses (< 1 year of age) should not be treated with ASIT yet.

ASIT treatment

Starter set:
The starter set contains 2 vials of allergen extract in 2 different concentrations (green and red cap) and will last for approx. 6 months of treatment.

Refill set:
The refill set contains only one vial with the higher concentration (red cap) and will last – depending on the individual injection interval – for about 10 months.

Cave:
It is not possible to perform hyposensitisation to food allergens!

Please enclose a veterinary prescription when ordering ASIT!
Delivery to the veterinary pharmacy will take 2 – 3 weeks.
Sending an invoice to the owner is not possible when ordering ASIT.

10. Drug Analysis / Intoxication

National and international anti-doping regulations in equestrian as well as in human competitive sports judge every qualitative detection of a substance (with few exceptions) as a positive result (so-called "zero option"). In equestrian sports, these regulations also apply to substances administered for therapeutic purposes. Yet, the subject of doping is in a continuous state of flux as the pharmaceutical industry on the one hand provides more and more agents, and on the other hand analytical procedures are permanently adapted and testing techniques are refined.
Which drug screening to choose for an individual horse depends on anamnesis, suspicious facts, exclusion attempts, etc. From our comprehensive Screening for Doping-relevant Substances up to a targeted analysis of a certain drug group, everything is possible.
Doping samples you send to LABOKLIN will be forwarded to an accredited partner laboratory for analysis. We will take care of the processing for you.

For the most frequent questions regarding drug level testing, we provide the following screenings. If you suspect the application of a specific medication, please note this on the submission form. Tests for drug residues not listed below are available on request. For special requests, please contact us by telephone.

10.1 Screening for Doping-relevant Substances

Testing for drugs and medicines in sport horses, also suitable as a pre-purchase examination.

Material: S 20 ml

Method: LCMS/MS + GCMS + CEDIA

Results: 2 – 3 weeks after receipt of sample. Express processing with findings after 3 working days is possible on request for an additional charge.

10.2 Antiphlogistics Screening

Analysis of glucocorticoids + NSAIDs + endogenous cortisol.

Material: S 20 ml

Method: LCMS/MS + GCMS

Results: see Chapter 10.1

10.3 Glucocorticoid Screening

Including endogenous cortisol

Material: S 10 ml

Method: LCMS/MS

Results: see Chapter 10.1

10.4 NSAID Screening

Material S 10 ml

Method GCMS

Results: see Chapter 10.1

10.5 Sedatives/Tranquiliser

Material: S 10 ml

Method: LCMS/MS

Results: see Chapter 10.1

10.6 Stimulants

Material: S 10 ml

Method: GCMS + CEDIA

Results: see Chapter 10.1

10.7 Tricyclic Antidepressants

Material: S 10 ml

Method: LCMS

Results: see Chapter 10.1

10.8 Meadow Saffron (Colchicine)

Colchicine is the main poison of Colchicum autumnale. Horses and other grazing animals can ingest the plant directly on the pasture or via hay or silage (colchicine is stable and remains toxic in hay and silage). Often, whole herds are affected. Possible signs of intoxication: colic, mucous-watery to bloody diarrhoea with secondary dehydration and electrolyte imbalances, central nervous manifestations such as apathy and unsteady gait, polyuria, haematuria, convulsions, respiratory depression, dyspnoea, cardiovascular failure, myopathies. Consequently, changes in the following laboratory parameters can occur: shift of electrolytes, initially peripheral leukocytosis, with progressing course leukopenia and pancytopenia, increase of liver and kidney values. The detection of critical amounts of poison can help to clarify the cause of the observed clinical signs.

Material: urine 1 ml

Results: 1 – 3 working days after sample receipt

Method: LCMS

Note:
Colchicine belongs to the category of doping-relevant substances in horses (FEI guidelines); traces may be detectable for up to several weeks after uptake.

10.9 Sycamore (Hypoglycin A)

Hypoglycine A (HGA) is a non-essential amino acid and can be found in various maple species, such as ash maple, sycamore maple, Japanese maple and others. It has not yet been detected in field and norway maple. The toxin is located especially in seeds and seedlings. This means that pasturing horses are particularly at risk of taking up toxic plant parts in spring (seedlings) and in autumn (seeds). Possible signs of HGA poisoning (equine atypical myopathy, sycamore poisoning) are apathy, ataxia, colic, lateral position, tremor, muscle weakness and pain, stiffness, discoloured urine, hyperthermia, dyspnoea up to respiratory arrest. Severe courses lead to rhabdomyolysis with myoglobinuria, colic and lying down. Finally the animals die from cardiac or respiratory arrest or from the secondary impact of rhabdomyolysis. Laboratory parameters that can give hints: severely elevated levels of CK, LDH, and AST.

Material: S 1 ml

Results: 1 – 3 working days after sample receipt

Method: LCMS

Note:
The detection of critical amounts of poison can help to clarify the cause of the observed clinical signs.

10.10 Ragwort (Senecionine)

Detection of the toxins senecionine and senecionine-N-oxide from urine.

Ragwort species (Senecio), especially the well-known Senecio jacobaea, are a common problem in meadows and pastures as they contain a large number of different pyrrolizidine alkaloids (PA). PAs are known as cumulative hepatotoxins, i.e. they cause damage to the genetic material of liver cells. This damage can accumulate over long periods of time. Acute poisoning caused by ingestion of large quantities is rather rare.

Unfortunately, the toxins also remain in hay and silage, and at the same time the plant parts lose bitter substances through storage, which increases the likelihood of ingestion.

The PA toxins senecionine and senecionine-N-oxide are the main toxins of ragwort and other native ragwort species in terms of quantity. These substances serve as marker toxins for the possible ingestion of poisonous plant parts. If senecionine and/or senecionine-N-oxide are detected in the urine of an animal, this indicates the oral ingestion of toxic plant parts within the last few hours to days. The substances do not naturally occur in the body.

Material: urine 1 ml

Method: LCMS

Results: 1 – 3 working days after sample receipt

11. Pathology

The normal processing time is 2 – 5 working days for cytological exams and 3 – 8 working days for histological specimens.

Informing about the anamnesis or the presumptive diagnosis is extremely important. This way, our pathologists' comments can be more case-specific and more beneficial for the colleague who ordered the examination.

11.1 Histopathology

11.1.1 Endometrial Biopsies

Endometrial biopsies can be taken from the mare at any stage of the oestrus cycle and sent fixed in formalin.
Endometrial biopsies are used to assess the probability of the mare getting in foal. Success of treatment (endometritis) can be monitored and irreversible degenerative changes and disturbances of differentiation in the endometrium can be identified.
The results of the histological examination (endometritis, endometrosis, angiosclerosis and state of endometrial differentiation) are categorised according to Kenney and Doig (1986). Diagnoses are further interpreted according to Schoon et al. (1992).
An endometrial biopsy can also be requested as a combined service together with breeding soundness examination and mycological examination.

11.1.2 Other Tissue Samples, e.g. Dermatopathology

Tumour samples, organ samples and skin biopsies should be sent fixed in formalin and, preferably, have a diameter of at least 5 mm. From this material, it is possible to do a histological and, where applicable, an immunohistological examination. For additional tests (e.g. microbiology), fresh material is required.

11.2 Cytology

11.2.1 Tracheobronchial Secretion (TBS) & Bronchoalveolar Lavage (BAL)

These tests are performed to assess the current state of lower airway disease. This is an important factor regarding prognosis and treatment options because differentiation between acute (especially bacterial) infection, allergic inflammation and chronic disease of the respiratory tract is possible. For evaluation purposes, it is important to know whether the sample is a lavage sample or directly obtained secretion and what kind of pre-treatment has taken place.
For cytological examinations, it is very important to prepare the smears directly after the sample collection to maintain adequate preservation of cellular detail. Otherwise, the sample will severely hampered by autolysis and bacterial overgrowth. In addition to the smear, the remaining fluid (cooled) and, if desired, a swab for bacteriological examination should be submitted.

11.2.2 Other Cytological Examinations

In horses, mainly synovia, abdominal/thoracic fluids and aspirates of cutaneous neoplasms are submitted for cytological examination. It is recommended to prepare smears immediately and submit them together with the remaining material (in an EDTA tube). Because of the low cell count, aqueous samples should be centrifuged (3 – 5 min, 2500 – 3000 rpm) before preparing a smear, and the smear should be prepared from the sediment. Please note on the submission form whether it is a sediment smear or a fresh. If an additional bacteriological examination is required, it is recommended to send in a swab.

12. Molecular Genetic Analysis

Hereditary Diseases/Coat Colours/Performance/Identity and Parentage
(For molecular genetic pathogen detection by PCR see Chapter 4, page 32 and Chapter 5, page 53.)

12.1 Hereditary Diseases

12.1.1 Androgen Insensitivity Syndrome (AR)
12.1.2 Cerebellar Abiotrophy (CA)
12.1.3 Congenital Stationary Night Blindness (CSNB2)*
12.1.4 Distichiasis*
12.1.5 Dwarfism
12.1.6 Dwarfism (ACAN, Chondrodysplasia)
12.1.7 Equine Juvenile Spinocerebellar Ataxia (EJSCA)*
12.1.8 Equine Malignant Hyperthermia (EMH)
12.1.9 Foal Immunodeficiency Syndrome (FIS)
12.1.10 Glycogen Branching Enzyme Deficiency (GBED)
12.1.11 Greying*
12.1.12 Hereditary Equine Regional Dermal Asthenia (HERDA)
12.1.13 Hereditary Myotonia
12.1.14 Hoof Wall Separation Disease (HWSD)
12.1.15 Hydrocephalus
12.1.16 Hyperkalaemic Periodic Paralysis (HYPP)
12.1.17 Idiopathic Hypocalcaemia
12.1.18 Immune-Mediated Myositis & MYH1 Myopathy (MYHM)
12.1.19 Incontinentia Pigmenti (Hyperpigmentation)
12.1.20 Junctional Epidermolysis Bullosa (JEB)
12.1.21 Lavender Foal Syndrome (LFS)
12.1.22 Naked Foal Syndrome (NFS)
12.1.23 Occipitoatlantoaxial Malformation (OAAM)*
12.1.24 Ocular Squamous Cell Carcinoma (SCC)
12.1.25 Overo Lethal White Syndrome (OLWS)
12.1.26 Polysaccharid Storage Myopathy Type 1 (PSSM)
12.1.27 Severe Combined Immunodeficiency (SCID)
12.1.28 Skeletal Atavism (SA)*
12.1.29 Splashed White (SW 1 – 8)
12.1.30 Warmblood Fragile Foal Syndrome (WFFS)

12.2 Coat Colours and Coat Structure

12.2.1 Agouti (black/bay)
12.2.2 Appaloosa Pattern 1
12.2.3 Brindle 1
12.2.4 Camarillo White – W4*

12.2.5 Champagne
12.2.6 Chestnut
12.2.7 Cream
12.2.8 Curly
12.2.9 Dominant White W5, W10, W13, W20, W22*
12.2.10 Dun
12.2.11 Greying*
12.2.12 Incontinentia Pigmenti (Hyperpigmentation)
12.2.13 Leopard Complex
12.2.14 Mushroom
12.2.15 Pearl
12.2.16 Roan Zygosity*
12.2.17 Sabino 1
12.2.18 Silver Dapple
12.2.19 Snowdrop
12.2.20 Splashed White (SW 1 – 8)
12.2.21 Sunshine
12.2.22 Tiger Eye*
12.2.23 Tobiano

12.3 Performance
12.3.1 Predictive Height Test
12.3.2 Speed Gene (Myostatin)*
12.3.3 SynchroGait (DMRT3)*
12.3.4 Tractability

12.4 Identity and Parentage
12.4.1 DNA Profile
12.4.2 Parentage

* = Partner laboratory

12.1 Hereditary Diseases

Variances or anomalies which have a genetic basis can already occur at birth or manifest themselves later in life.
Most congenital defects are actually not hereditary.
As research into the equine genome progresses, molecular biological studies could detect the genetic fixation of some of these diseases, though.

Below, we will describe breed dependencies, clinical picture, inheritance and diagnosis of the defects which are proven to be genetically fixed.

To perform a genetic test, an **EDTA whole blood sample** (approx. 0.5 ml) is required. In horses, it is also possible to send in **hairs with roots** (approx. 20 hairs, plucked from mane or tail), however, the amount of DNA which can be extracted from hairs is limited. **Important: For all partner laboratory tests (except speed gene)**, hair with **hair roots** is required as sample material.

DNA extracted for genetic tests is **stored** at LABOKLIN for at least 5 years. This DNA can then be used for future genetic tests or paternity tests. In most cases, it is therefore not necessary to send in a new sample.

12.1.1 Androgen Insensitivity Syndrome (AR)

AR1

Method	sequencing
Breed	Quarter Horse and related breeds
Inheritance	x-chromosomal recessive
Test duration	1 – 2 weeks after sample receipt

AR2, AR3, AR4, AR5*

Method	partner laboratory
Breed	Tennessee Walking Horse, Thoroughbred, Warmblood
Inheritance	x-chromosomal recessive
Duration	4 – 6 weeks after sample receipt

Disease

In mammals, gender is determined by the X and Y chromosomes. They are responsible for numerous factors that determine the proper development of the sexual phenotype. The role of androgens is crucial for normal male sex differentiation. The intracellular androgenic effect is mediated by the androgen receptor (AR). If its function is impaired, the cells in the body are not able to respond to the androgen. This leads to a variety of syndromes with severe clinical consequences, most notably androgen insensitivity syndrome (AR): XY (genetically male) horses have a female phenotype (female external genitalia) and internal testicles. These horses often behave like stallions, but are not capable of reproduction.
Affected breeds are Quarter Horses and related breeds as well as Tennesse Walking Horses und Warmbloods.

12.1.2 Cerebellar Abiotrophy (CA)

Method	TaqMan SNP assay
Breed	Arabian Horses and their crossbreeds
Inheritance	autosomal recessive
Duration	3 – 5 working days after sample receipt

Disease
CA is a neurological disease which almost exclusively affects Arabian Horses and their crossbreeds. Affected foals are usually born with no symptoms; already in the first weeks of life, the disease causes the death of neurons in the cerebellum. This leads to neurological deficits such as head shaking, ataxia and other deficiencies. The first signs usually appear at the age of 6 weeks (up to 4 months). The neurological problems may not be apparent to owners and are frequently thought to be a consequence of a fall/an accident rather than CA.
The severity of the symptoms of CA is variable. Some foals show very severe signs, including, most notably, exaggerated gait and lack of balance. Other animals only show mild clinical signs, but in almost all cases it is impossible to ride these horses later.

12.1.3 Congenital Stationary Night Blindness (CSNB2)*

Method	partner laboratory
Breed	Missouri Fox Trotter, Standardbred, Tennessee Walking Horse
Inheritance	autosomal recessive
Duration	4 – 6 weeks after sample receipt

Disease
In congenital stationary night blindness (CSNB), affected animals cannot see in low light or darkness. CSNB is not progressive. Some typical signs of CSNB are fear of unfamiliar places in the dark, difficulty finding food or water buckets at night, or being prone to injuries at night-time. CSNB in horses is often not detected by the owner. The definitive diagnosis of CSNB is made by electroretinogram.
Similar to humans and other animals, there are probably several different genes that contribute to this disease in horses, and these genes are thought to be breed-specific. Based on population screening, it is estimated that one in one hundred Tennessee Walking Horses is homozygous for this variant and therefore likely to be night blind. Congenital stationary night blindness can also be caused by a homozygous mutation in the leopard gene. To test for the presence of this mutation, the Leopard Complex test should be requested (see Chapter 12.2.13, page 108).

12.1.4 Distichiasis*

Method	partner laboratory
Breed	Friesian
Inheritance	autosomal recessive with incomplete penetrance
Duration	4 – 6 weeks after sample receipt

Disease
Abnormal growth of eyelashes from the Meibomian glands leads to misplaced eyelashes. They can cause irritation and inflammation of the cornea, excessive tearing, squinting and pain up to ulceration and scarring of the cornea. Loss of vision may occur

or the eye may need to be removed. In some horses, there are no signs of abnormal growth of the eyelashes, so it may remain undetected that these horses will transmit this genetic variant in any case.

12.1.5 Dwarfism

Method	sequencing
Breed	Friesian
Inheritance	autosomal recessive
Duration	1 – 2 weeks after sample receipt

Disease
Dwarfism in Friesian horses is characterised by growth retardation of the ribs and limbs, while the head and back appear normal. A striking feature of the condition is hyperextension of the fetlock joints. The flexor tendon does not contract as usual when the foals grow older, but tends to stretch further. As a consequence, these dwarf Friesians develop an abnormal gait in which the limbs undergo extreme rotation at the carpus and hocks. The head in adult dwarf Friesians is the same size as in healthy animals, the ribcage is wider than normal with a thickening of the costochondral junction (Th10-16). The back appears disproportionally long, but the legs are greatly shortened. The abdomen is usually rounded, the muscles of the whole body are only poorly developed. Dwarfism in Friesian horses is caused by a mutation in the B4GALT7 gene.

12.1.6 Dwarfism (ACAN, Chondrodysplasia)

Method	sequencing
Breed	American Miniature Horse, Shetland Pony
Inheritance	see disease
Duration	1 – 2 weeks after sample receipt

Disease
Dwarfism is most common in Shetland ponies and miniature horses. Phenotypical features of this hereditary disease are breathing problems due to a cleft palate, deformed mouths, shortened limbs and bowed forelegs, disproportionately large cranium and shortened neck, protruding eyes, abdominal hernia and a shortened rib cage. As a result, affected animals are often not viable or have to be euthanized due to the poor quality of life. A mutation in the ACAN gene is responsible for this form of dwarfism. Four different mutations, which cause the autosomal-recessive disease, are known so far. These are named D1, D2, D3*, and D4 and are also pathogenic as compound heterozygous genes. Compound heterozygous variants together with D1 (except N/D1) are more deleterious and often lead to death. A combination with the D2 variant is considered as the mildest form of dwarfism.

12.1.7 Equine Juvenile Spinocerebellar Ataxia (EJSCA)*

Method	partner laboratory
Breed	Quarter Horse
Inheritance	autosomal recessive
Duration:	4 – 6 weeks after sample receipt

Disease
In 2020, a new neurological disease was identified in the American Quarter Horse, equine juvenile spinocerebellar ataxia (EJSCA). Affected foals show coordination problems or ataxia between the first and fourth week of life. Within a few days, the foals are no longer able to stand independently and have to be euthanised. Blood analyses show elevated glucose and gamma-glutamyltransferase levels. Post-mortem examinations showed pronounced lesions in the spinal cord. The genetic variant underlying this disease has been identified, but has not yet been published.

12.1.8 Equine Malignant Hyperthermia (EMH)

Method	TaqMan SNP assay
Breed	all
Inheritance	autosomal dominant
Duration	3 – 5 working days after sample receipt

Disease
Equine malignant hyperthermia (EMH) is an inherited disorder of skeletal muscle characterised by hyperthermia, metabolic acidosis, rhabdomyolysis, generalised spasms of the skeletal muscles, cardiac arrhythmia and renal dysfunction. The clinical signs appear after exposure to muscle relaxants, halothane anaesthesia or stress. EMH appears to increase the symptoms of PSSM. It especially affects Quarter Horses and related breeds.

12.1.9 Foal Immunodeficiency Syndrome (FIS)

Method	sequencing
Breed	Dales Pony, Fell Pony, Tinker
Inheritance	autosomal recessive
Duration	1 – 2 weeks after sample receipt

Disease
Foal immunodeficiency syndrome (FIS) is a hereditary disease that, so far, has only been detected in Fell Ponies and Dales Ponies. The gene variant has also already found in the Tinker. Foals with FIS appear to be normal at birth but as they lack immunity, they develop a number of diseases, especially pneumonia and diarrhoea at a few weeks of age. The foals also suffer from severe progressive anaemia and usually die before 3 months of age.

12.1.10 Glycogen Branching Enzyme Deficiency (GBED)

Method	TaqMan SNP assay
Breed	Appaloosa, Paint Horse, Quarter Horse and related breeds
Inheritance	autosomal recessive
Duration	3 – 5 working days after sample receipt

Disease

Until recently, GBED had not been recognised as a disease, mainly because the clinical signs are very similar to other diseases that typically affect foals. Furthermore, routine staining of muscle tissue post mortem was not suitable to detect GBED.
Since molecular biology was able to detect a genetic defect and provide a genetic test, epidemiological studies have revealed that about 10 % of the horses belonging to Quarter Horse, Paints and related bloodlines carry the mutation. It is assumed that GBED is responsible for at least 3 % of the abortions in Quarter Horses.
Affected foals lack the enzyme GBE that is necessary for synthesis and storage of glycogen. Predominantly affected tissues are skeletal muscles, brain and heart.
Clinical signs associated with GBED are:

- abortion, stillbirth or birth of weak foals
- sudden cardiac death – especially on the pasture – or death caused by seizures
- tachypnoea due to weakened respiratory muscles
- generalised weakness, especially when getting up

All cases known so far were euthanised or died at the latest by the age of 18 weeks.

12.1.11 Greying*

Method	partner laboratory
Breed	all
Inheritance	autosomal dominant
Duration	4 – 6 weeks after sample receipt

Phenotype / Disease

Horses carrying the greying mutation are born coloured and gradually lose the pigmentation of their hair. The first signs of grey hair are usually found on the head, especially around the eyes. The pigmentation of the skin remains in its original colour. Horses become completely grey/white by the age of 6 – 12 years.
The genetic cause of this is a gene variant in the STX17 gene. There are three alleles: N = normal or not grey, G2 = greying gene duplication, two tandem copies of the duplicated sequence cause grey/white, G3 = grey gene triplication, three tandem copies of the sequence also cause grey/white, but these horses turn grey much faster and are often already completely white at a few years of age.
Greying is inherited autosomal-dominantly, i.e. even a single copy of one of the two alleles G2 or G3 causes a horse to turn grey.

The formation of melanomas is directly linked to the greying mutation, with 70 – 80 % of all grey horses over the age of 15 having one or more melanomas. Horses with the G3 variant grey faster and have a higher risk of melanoma than those with G2.
The probability of this (incidence) is also statistically dependent on the basic colour of the horse, which is determined by another DNA section, the agouti locus. Grey horses born black have a significantly higher risk of developing melanoma than white horses born brown. It is also reported that grey horses have a higher risk of squamous cell carcinoma of the eye than other horses.
A 2024 study led by Dr Leif Andersson and colleagues at Uppsala University in collaboration with researchers at the UC Davis Veterinary Genetics Laboratory (VGL) showed that the speed of greying and the risk of melanoma are influenced by the number of copies of this sequence responsible for greying in horses.
It was shown that horses without duplication (N/N) do not grey and have the lowest incidence of melanoma. Horses with a G3 copy (N/G3) greyed faster and melanomas occurred more frequently, while horses with a homozygous G3 copy (G3/G3) greyed fastest and developed melanomas most frequently. Horses with the G2 allele, whether heterozygous (N/G2) or homozygous (G2/G2), showed a low incidence of melanoma, similar to horses without duplications (N/N).

12.1.12 Hereditary Equine Regional Dermal Asthenia (HERDA)

Method	TaqMan SNP assay
Breed	Appaloosa, Paint Horse, Quarter Horse and related breeds
Inheritance	autosomal recessive
Duration	3 – 5 working days after sample receipt

Disease
Hereditary equine regional dermal asthenia (HERDA) is a degenerative skin disease affecting predominantly American Quarter Horses. Within the population, the number of carriers of this disease is about 1.8 – 6.5 %.
Normally, foals are born without any signs of the disease. Skin areas that later develop lesions are focal and irregularly distributed all over the body with a predilection for the area on the back. Consequently, the disease is often only discovered when the horses are saddle broken – at about 2 years of age.
The skin of affected horses is hyper-extensible, scarred, and often shows severe lesions. Histological examinations of skin biopsies are not suitable for diagnosis of HERDA and can only provide hints.

12.1.13 Hereditary Myotonia

Method	TaqMan SNP assay, if necessary sequencing
Breed	New Forest Pony
Inheritance	autosomal recessive
Duration	3 – 5 working days after sample receipt (1 – 2 weeks for sequencing)

Disease
Congenital myotonia is a disease of the skeletal muscles. It is caused by a mutation in the CLCN1 gene, which is responsible for the function of chloride channels in the muscles.
The first signs of the disease already appear at a few weeks of age. Foals have a stiff-legged gait, are recumbent and have considerable difficulty getting back on their feet after a long period of lying. In most cases, it is impossible to lift one single foot, the foals lose their balance very quickly. The eyeballs may also be retracted deeply into the eye sockets due to myotonia.

12.1.14 Hoof Wall Separation Disease (HWSD)

Method	TaqMan SNP assay
Breed	American Miniature Horse, Connemara Pony, German Riding Pony
Inheritance	autosomal recessive
Duration	3 – 5 working days after sample receipt

Disease
Hoof wall separation disease (HWSD) is characterised by a very unstable hoof wall which can crack and break without any particular strain. The coronary band appears normal. Symptoms already appear in the first weeks of life and can be of varying severity. However, these horses are usually not suited for riding later.

12.1.15 Hydrocephalus

Method	sequencing
Breed	Friesian
Inheritance	autosomal recessive
Duration	1 – 2 weeks after sample receipt

Disease
Hydrocephalus in Friesian horses is a developmental disorder that can lead to a progressive enlargement of the head. It has been associated with dystocia in dams and stillbirths of the affected foals. It can eventually lead to fatal complications for the dam at parturition. Affected foals that are still alive may be euthanised at birth to facilitate parturition.
Hydrocephalus in Friesian horses is caused by a mutation in the B3GALNT2 gene.

12.1.16 Hyperkalaemic Periodic Paralysis (HYPP)

Method	TaqMan SNP assay
Breed	Appaloosa, Paint Horse, Quarter Horse and related breeds
Inheritance	autosomal dominant
Duration	3 – 5 working days after sample receipt

Disease
Hyperkalaemic periodic paralysis (HYPP) has been reported in Quarter Horses (QH), Paints, Appaloosas and other bloodlines descending from the QH stallion "Impressive". Most of the affected horses are very well-muscled and may be very successful sport or show horses between episodes of illness.
The main clinical signs is general weakness; muscle spasms and fasciculations may occur. The manifestation of the clinical signs ranges from subclinical to severe. Life-threatening complications are cardiac arrhythmia and risk of suffocation caused by laryngospasm. When clinical signs occur, hyperkalaemia can be found by laboratory diagnostics; muscle values are usually within the reference range or slightly above.
The first episodes of clinical illness are usually observed at the age of 3 – 7 years. HYPP is caused by a mutation of a base in the gene that encodes for the sodium channels of skeletal muscles. It is inherited as an autosomal dominant trait. Homozygous carriers of the defect gene become more severely ill than heterozygous animals.
In contrast to sporadic exertional rhabdomyolysis, which is always associated with movement in horses, HYPP usually does not occur in connection with exercise, but during periods of rest, at feeding time or in stressful situations (transport, change of food, fasting); periods of standing and a diet rich in K can also trigger clinical signs.

12.1.17 Idiopathic Hypocalcaemia

Method	sequencing
Breed	Thoroughbred
Inheritance	autosomal recessive
Duration	1 – 2 weeks after sample receipt

Disease
A lethal idiopathic hypocalcaemia was described in 1997 for thoroughbred foals. The affected foals suffer from muscle spasms and seizures within the first weeks of life due to calcium deficiency. Other manifestations can be a stiff gait and increased sweating. The foals died within a few weeks or were euthanized due to the poor prognosis.
In addition to calcium deficiency, the blood count also shows a magnesium deficiency and an increased phosphate level. The parathormone (PTH) increases normally with calcium deficiency. However, no increased PTH concentration was found in the affected foals.
In 2020, the genetic cause underlying calcium deficiency could be described. A gene variant in the RAPGEF5 gene is associated with hypoparathyroidism. This subfunction in turn causes a reduced PTH production, which causes the calcium deficiency.
Only animals carrying two copies of the disease-causing gene variant show symptoms. The gene variant has so far only been described in English thoroughbreds. However, since thoroughbreds are used in the breeding of other breeds, further distribution cannot be excluded.

12.1.18 Immune-Mediated Myositis & MYH1 Myopathy (MYHM)

Method	sequencing
Breed	Appaloosa, Paint Horse, Quarter Horse and related breeds
Inheritance	autosomal dominant with variable penetrance
Duration	1 – 2 weeks after sample receipt

Disease

Appaloosas, Paint Horses and Quarter Horses are susceptible to an autoimmune muscle disease called immune-mediated myositis (IMM) which can cause severe atrophy, resulting in the loss of up to 40 % of muscle mass within 72 hours. IMM is characterised by the infiltration of inflammatory cells, especially lymphocytes, into muscle fibres und surrounding blood vessels, which manifests itself in stiffness, weakness and non-specific malaise. Affected horses are usually 8 years and younger or 17 years and older. Environmental factors combined with genetic susceptibility are important triggers for the development of muscle atrophy or severe rhabdomyolysis. About 39 % of the horses with IMM have already been suffering from a triggering factor for some time such as an infection with Streptococcus equi subsp. equi or EHV4.

A variant in the MYH1 gene which inhibits the function of the myosin protein in muscle cells, is associated with increased susceptibility to develop IMM. Another clinical presentation of the MYH1 variant in young Quarter Horses is severe, sudden muscle damage which occurs without any physical exercise and is not necessarily associated with muscle atrophy (non-exertional rhabdomyolysis). IMM and non-exertional rhabdomyolysis belong to the group of muscle diseases known as MYH1 myopathy (MYHM).

The mode of inheritance for MYHM is autosomal dominant with incomplete penetrance, which means that not all horses that have one (genotype N/My) or two alleles (genotype My/My) of the genetic variant will develop IMM or non-exertional rhabdomyolysis. Horses with two copies (My/My) may be more severely affected.

12.1.19 Incontinentia Pigmenti (Hyperpigmentation)

Method	sequencing
Breed	all
Inheritance	x-chromosomal incomplete dominant
Duration	1 – 2 weeks after sample receipt

Phenotype / Disease

Incontinentia pigmenti (IP) is an ectodermal dysplasia in Quarter Horses and related breeds. It is characterised by skin lesions evolving over time, as well as dental, nail and ocular abnormalities.

Soon after birth, pruritic, exudative lesions occur in affected horses, sometimes developing into verrucous lesions. There may be areas of alopecia with occasional woolly hair re-growth. Affected horses also show streaks of darker and lighter coat colour from birth, similar to brindle coat colour. Due to the X-linked dominant inheritance, IP signs can only be seen in mares while affected male embroys die in utero.

12.1.20 Junctional Epidermolysis Bullosa (JEB)

JEB1

Method	sequencing
Breed	Belgian Draft
Inheritance	autosomal recessive
Test duration	1 – 2 weeks after sample receipt

JEB 2*

Method	partner laboratory
Breed	American Saddlebred
Inheritance	autosomal recessive
Duration	4 – 6 weeks after sample receipt

Disease

Junctional epidermolysis bullosa (JEB) is an inherited disease of the skin which mainly affects Belgian Draft Horses but can also be found in American Saddlebred Horses. The mode of inheritance is autosomal recessive and causes blistering and detaching of the skin. Affected foals are already born with lesions or develop them after only 2 days at the latest. Primary skin lesions are vesicles that rupture easily and become sharply contoured ulcers with exudation and encrustation. Predilection sites are the coronary band, where changes up to sloughing may occur, mucocutaneous junctions of the lips, anus, vulva, eyelids and nostrils; in addition, all skin areas covering prominent bones are affected (fetlock, carpus, hips, etc.). Another sign is dental dysplasia. The foals eventually have to be euthanised due to infections.

12.1.21 Lavender Foal Syndrome (LFS)

Method	TaqMan SNP assay
Breed	Arabian Horses and their crossbreeds
Inheritance	autosomal recessive
Duration	3 – 5 working days after sample receipt

Disease

Lavender foal syndrome (LFS) is an autosomal recessive defect which occurs in a subgroup of the Arabian thoroughbred, the Egyptian Arabian. Affected foals show a range of neurological signs, including convulsive seizures, opisthotonus or nystagmus. They are normally unable to stand and nurse from their mother and are usually euthanised if they do not die immediately after birth.
The name "Lavender Foal Syndrome" originates from the fact that the causative gene for LFS is linked to another gene that is responsible for the dilution factor "lavender". That is why affected foals often show the characteristic "lavender" coat colour.

Note:
Not every foal born with a "lavender"-coloured coat is necessarily a (homozygous) carrier of the mutation.

12.1.22 Naked Foal Syndrome (NFS)

Method	TaqMan SNP assay
Breed	Akhal-Teke
Inheritance	autosomal recessive
Duration	3 – 5 working days after sample receipt

Disease

NFS is a genodermatosis in the Akhal-Teke horse breed. Affected horses are born with almost no hair. They show a mild form of ichthyosis and mostly die within days to weeks after birth. So far, the reason for the early death is not known; only few hairless foals have survived up to 2.5 years. The first records of hairless Akhal-Teke foals date back to 1938, and since then, the number of such foals has been steadily increasing. Many horses with NFS were probably registered as stillborn or not registered at all.

12.1.23 Occipitoatlantoaxial Malformation* (OAAM)

Method	partner laboratory
Breed	Arabian
Inheritance	autosomal recessive
Duration	4 – 6 weeks after sample receipt

Disease

OAAM is characterised by a fusion of the occipital bone with the atlas. An additional malformation of the axis with an accompanying shortened dens can cause an unstable connection between the atlas and axis. Subluxation of the atlantoaxial joint is also possible. The resulting compression of the spinal cord can cause neurological signs. Affected horses show an abnormal head and neck posture and reluctance to move the neck. Clinical signs range from weakness of the limbs to progressive ataxia.
OAAM is inherited as an autosomal recessive genetic defect in Arabian horses. Several mutations appear to be causative.
One of these variants was identified by researchers at the School of Veterinary Medicine, University of California, Davis. This variant, which is associated with a form of OAAM in Arabians, consists of a large deletion in the homeobox gene cluster (HOX). Other genetic bases of other forms of OAAM are the subject of current research.

12.1.24 Ocular Squamous Cell Carcinoma (SCC)

Method	sequencing
Breed	Belgian Draft (Ardennais, Brabant), Belgian Warmblood, Connemara Pony, Haflinger, Rocky Mountain Horse
Inheritance	autosomal recessive
Duration	1 – 2 weeks after sample receipt

Disease
Squamous cell carcinoma (SCC) is the second most common type of tumour in the horse and the most frequent tumour in the horse's eye. Factors thought to increase risk of SCC include UV exposure, pigmentation and genetic factors. As a risk factor for the development of SCC on the limbus or on the third eyelid, a variant in the DDB2 gene in Haflingers, Ardennes Horses and Belgian Drafts was identified.
When originating at the limbus, SCC can spread into the cornea and quickly lead to visual impairment and destruction of the eye.
Horses homozygous (R/R) for the risk factor are 5.6 times (Haflinger) or 4.0 times (Belgians) more likely to develop ocular SCC than those with one copy (R/N) or no copies (N/N) of the risk factor. This risk factor does not explain all cases of ocular SCC but it appears to be a major contributor in Haflingers and Belgians. Furthermore, the homozygous genotype was found in horses of the breeds Rocky Mountain Horse, Connemara Pony and in a Holsteiner/Belgian Warmblood mix, which exhibited squamous cell carcinoma of the eye.
Homozygous horses (R/R) are advised to have routine eye exams for early detection and better prognosis, and to wear a UV protection fly mask when out during the daylight hours.

12.1.25 Overo Lethal White Syndrome (OLWS)

Method	TaqMan SNP assay
Breed	Appalosa, Paint Horse, Quarter Horse and other breeds with overo pattern
Inheritance	autosomal recessive
Duration	3 – 5 working days after sample receipt

Disease
OLWS is a lethal autosomal recessive defect which mainly occurs when mating frame overo Paints. However, also miniature horses, Arabian crossbreeds, thoroughbreds, Quarter Horses, Mustangs as well as Tobiano Paint Horses without the typical coat pattern can be carriers of the mutation of the endothelin-B receptor gene.
Affected foals are born completely white and show intestinal aganglionosis. The foals develop severe colic due to the resulting functional ileus and normally die within 24 to 48 hours.
Note:
Not every foal of the respective breeds which is born white is necessarily a (homozygous) carrier of the mutation.

12.1.26 Polysaccharid Storage Myopathy Type 1 (PSSM)

Method	TaqMan SNP assay
Breed	all
Inheritance	autosomal dominant
Duration	3 – 5 working days after sample receipt

Disease
Polysaccharide storage myopathy (PSSM) is a glycogen storage disease which weakens the affected horses and may even be life-threatening. Affected breeds are mainly Quarter Horses, American Paints, Appaloosas but also draft horses, warmbloods, ponies and crossbreeds of all breeds mentioned above.
PSSM is characterised by the accumulation of abnormal polysaccharides and the excessive accumulation of the normal form of sugar in muscle tissue.
The clinical signs are similar to those of sporadic exertional rhabdomyolysis and include the entire spectrum from reluctance to move to muscle tremor, muscle stiffness, sweating, alternating lameness, stretching of the hind legs up to immobility. Episodes usually begin after 10 – 20 minutes of light exercise. The muscles of the mainly affected hind legs are often hard or even painful.
Many horses with PSSM have a history of numerous episodes of muscle problems. If the clinical picture is pronounced, myoglobinuria and, possibly, resulting renal impairment may occur. Generally, PSSM is held responsible for the majority of neuromuscular diseases in the affected breeds.
PSSM is inherited in an autosomal dominant mode, which means that already one copy of the affected allele can cause the disease. Horses that are homozygous for the mutation are often more severely affected.
Laboklin owns the exclusive license to perform this genetic test.

12.1.27 Severe Combined Immunodeficiency (SCID)

Method	fragment length polymorphism
Breed	Arabian
Inheritance	autosomal recessive
Duration	1 – 2 weeks after sample receipt

Disease
Severe combined immunodeficiency (SCID) is the oldest known hereditary disease in horses. Affected breeds are Arabian Horses and their crossbreeds. It is a primary, lethal immunodeficiency characterised by the inability of the horse to produce B and T lymphocytes. Gamma interferon and IgM are also deficient. The inheritance is autosomal recessive, the incidence is 2 – 3 % with a carrier frequency of about 25 %. Affected foals are extremely susceptible to infections. Depending on the supply of maternal antibodies, sooner or later (at the latest at 2 months of age), the foals will suffer from opportunistic pathogens, typically also from adenovirus infections. The respiratory and the gastrointestinal tract are particularly affected. In addition, high-grade persistent lymphopenia occurs. Most of the affected foals die before they are 5 months old.

12.1.28 Skeletal Atavism (SA)*

Method	partner laboratory
Breed	American Miniature Horse, Shetland Pony

Inheritance	autosomal recessive
Duration	4 – 6 weeks after sample receipt

Disease

Skeletal atavism is characterised by the ulna and fibula growing too long and failing to fuse with the radius and tibia, respectively. This results in severe angle anomalies and deformation of the carpal joints and hock joints, typically short limbs, a low rectangular body shape, abnormal limb position and movement disorders. The angles of the limbs and the movement pattern become more abnormal as the foal ages and in most cases the horse has to be euthanised within six months.
A Swedish research team has identified two independent overlapping regions in the SHOX gene in which DNA sequences were lost (deletions) in affected ponies. The deletions (Del1 and Del2) vary in size, with the larger deletion (Del1) occurring more frequently in ponies. It is estimated that around 12 % of Shetland Ponies are carriers.

12.1.29 Splashed White (SW 1 – 8)

Horses with splashed white piebalding can be deaf.
Splashed white is an uneven piebald pattern characterised by an extremely wide blaze or lantern, often in combination with blue eyes, and high legs. So far, 8 causative mutations have been identified (SW 1 – SW 8). Further information can be found in Chapter 12.2.20, page 111.

12.1.30 Warmblood Fragile Foal Syndrome (WFFS)

Method	TaqMan SNP assay
Breed	Warmblood, Appaloosa, Haflinger, Mustang, Paint Horse, Quarter Horse
Inheritance	autosomal recessive
Duration	3 – 5 working days after sample receipt

Disease

Warmblood fragile foal syndrome (WFFS) is an inherited connective tissue disorder that becomes apparent immediately after the foal is born. Symptoms are comparable to Ehlers-Danlos-Syndrome in humans. The skin is extremely fragile and tears at even the slightest touch. In addition to numerous injuries all over the body and enlargements of the joints (joint effusion), gingiva and mucous membranes can also be affected. Limb joints are hyperextensible, most clearly visible at the fetlock joints. That is why affected foals are mostly unable to stand properly. As the prognosis is very poor, foals with WFFS are euthanised shortly after birth.
Not all foals are born after a normal period of gestation, premature births and abortions due to WFFS are also known.
Laboklin owns the exclusive license to perform this genetic test (Europe).

12.2 Coat Colour and Coat Structure

Horses have 2 main types of skin pigments that are responsible for the coat colour. One base colour is dark (black or brown/bay) and the other one is red. The many colours and shades in between are the result of genes that regulate the amount, strength and distribution of the 2 main pigment types.
For information on sample material and DNA storage see Chapter 12.1, page 87.

12.2.1 Agouti (black/bay)

Method fragment length polymorphism
Breed all
Duration 1 – 2 weeks after sample receipt

Phenotype
The distribution of the black pigment is controlled by the Agouti (A) locus. In chestnuts, this gene does not play a role in determining the coat colour. A black or brown/bay coat colour can only develop if a horse is either a non-carrier (E/E) or only a carrier (E/e) of the chestnut coat colour. The Agouti gene determines whether the horse develops a black or a brown/bay coat. If the recessive allele is homozygous (a/a), the horse will be black. Otherwise (carrier: A/a or non-carrier: A/A), the coat is brown/bay and black appears only in certain areas like mane or tail.
For breeding purposes, the Agouti gene is particularly important as a "hidden" feature in chestnut horses.

Allele Combination	Agouti A/A	Agouti A/a	Agouti a/a
Extension E/E	brown/bay	brown/bay	black
Extension E/e	brown/bay	brown/bay	black
Extension e/e	chestnut	chestnut	chestnut

12.2.2 Appaloosa Pattern 1

Method TaqMan SNP assay
Breed all
Duration 3 – 5 working days after sample receipt

Phenotype
While the gene for the Leopard complex (LP) is responsible for various white patterns, the PATN1 gene determines the extent and distribution of white parts. A mutation in the PATN1 gene is associated with an increased amount of white in in LP horses. In horses that are heterozygous for LP (LP/lp), the presence of the PATN1 mutation usually produces a leopard pattern. In horses that are homozygous for LP (LP/LP), the PATN1 mutation most often results in a few spot pattern. The PATN1 mutation is often present

in breeds with LP, including Appaloosa, British Spotted Pony, American Miniature Horse and Knabstrupper. It has been found in other breeds, too, but has no effect on the phenotype without the LP gene.

12.2.3 Brindle 1

Method	sequencing
Breed	all
Duration	1 – 2 weeks after sample receipt

Phenotype
The phenotype "brindle" shows irregular vertical stripes in the coat along the neck, back and hind legs. This specific form of brindle is called "brindle 1 (BR1)". In some BR1 horses, the stripes are differently pigmented. Most horses show a sparse mane and tail. BR1 has an X-chromosomal semi-dominant mode of inheritance. The typical BR1 phenotype with a brindle coat pattern can only be seen in heterozygous females (genotype X(N)/X(BR1)), whereas homozygous females (genotype X(BR1)/X(BR1)) and hemizygous males (genotype X(BR1)/Y) show a sparse mane and tail but no stripes.

12.2.4 Camarillo White W4*

Method	partner laboratory
Breed	all
Duration	4 – 6 weeks after sample receipt

Phenotype
The W4 mutation in the KIT gene is a mutation which is traced back to the stallion "Sultan". He was born in 1912 and Adolfo Camarillo bred him to Morgan Horse mares. These very successful white horses were bred by the Camarillo family until 1987 when they were sold at an auction all over the world.
The genetic test enables breeders who would like to breed this colour to find out whether or not a horse that is a descendant of Sultan and only has few white markings itself, can pass on the mutation.
The genotype W4/W4 has not yet been observed. It is therefore likely that homozygous horses die in utero.

12.2.5 Champagne

Method	TaqMan SNP assay
Breed	all
Duration	3 – 5 working days after sample receipt

Phenotype
Similar to the cream gene, the champagne gene causes a dilution of the base colour. It is inherited in a dominant manner and the difference in phenotype between homozygous (CH/CH) and heterozygous (CH/ch) carriers can hardly be distinguished.

The base colour chestnut is diluted to gold champagne, bay becomes amber champagne and black becomes classic champagne.
Champagne horses are born with pink skin that develops freckles during the first days of life. In most cases, the eyes are blue but darken as the horse gets older.

12.2.6 Chestnut

Method	TaqMan SNP assay
Breed	all
Duration	3 – 5 working days after sample receipt

Phenotype
The Extension (E) locus determines the red factor (chestnut). The dominant allele (E) leads to the formation of eumelanin and thus to the colour bay or black. The disposition for chestnut coat colour (e) is inherited in a recessive manner, i.e. only if the mutation in the McR1 gene is homozygous (e/e), phaeomelanin will be produced and the horse will be chestnut. A horse that is homozygous (E/E) or heterozygous at the E-locus (E/e) will be either black or bay, depending on the Agouti locus.

12.2.7 Cream

Method	TaqMan SNP assay
Breed	all
Duration	3 – 5 working days after sample receipt

Phenotype
Another gene, MATP, determines the different variations of the coat colour cream. Depending on the base colour, which is determined by the genes of the E- and the A-locus, this gene can create the following colour shades:
Non-carriers of the gene (cr/cr) have the base colour chestnut, bay or black.
Chestnut carriers (e/e; CR/cr) are diluted to palomino (isabelline), chestnut homozygous carriers (e/e; CR/CR) become cremello.
Bay carriers (E/e or E/E; A/A or A/a; CR/cr) become buckskin, bay homozygous carriers (E/e or E/E; A/A or A/a; CR/CR) are diluted to perlino.
Black carriers (E/e or E/E; a/a; CR/cr) become smoky black, black homozygous carriers (E/e or E/E; a/a; CR/CR) become smoky cream.
Our test specifically detects a mutation in the MATP gene, which has been shown to be responsible for the described cream colours. Other genes or mutations causing similar coat colours are not identified by this test.

12.2.8 Curly

Method	TaqMan SNP assay
Breed	all
Duration	3 – 5 working days after sample receipt

Phenotype
Curly coat is a special trait in horses and leads to a curly coat structure. It occurs mainly in the American Bashkir Curly Horse, but it can be found in various other horse breeds, too. Nowadays, Curly horses are very popular because their coat structure causes milder or no symptoms at all in many horse-allergic people.
The curly coat structure sometimes appears in combination with hypotrichiosis.
Genetic variants in two different genes, KRT25 and SP6, are responsible for the development of the curly coat and hypotrichiosis. Horses that are heterozygous or homozygous for only the KRT25 variant, show curly coat and hypotrichiosis, while horses with only the SP6 variant have curly hair without hypotrichiosis. Horses with mutated alleles in both variants develop curly hair and hypotrichiosis. All horses with the KRT25 variant are hypotrichiotic because of the epistatic effect that KRT25 has on SP6. In our genetic test, both variants are examined separately.

12.2.9 Dominant White W5, W10, W13, W20, W22*

Method	partner laboratory
Breed	all
Duration	4 – 6 weeks after sample receipt

Phenotype
The KIT gene has a crucial function in the development of many cell types, including blood and pigment cells (melanocytes). Mutations that affect the normal function of the KIT protein often lead to a lack of melanocytes in the skin and hair follicles, resulting in a white pattern in horses known as dominant white.
The dominant white pattern is variable and ranges from extensive facial and leg markings with or without minimal sabino-like patterns, including roaning on the abdomen and/or abdomen patches, to a pure white horse. The eye colour of dominant white horses is typically brown.
A number of different KIT mutations associated with white patterns have been identified in the horse. These include dominant white, sabino-1 and tobiano. To date, 34 of these mutations have been characterised as dominant white mutations and range from variants that have a minimal effect on coat patterning to those that cause a pure white phenotype.
Many of the dominant white mutations have arisen recently and are therefore restricted to certain lines within the breeds. Among the exceptions are W13 and W20.

W13 was originally identified in Quarter Horses, but has also been described in several other breeds, including the Australian Miniature Horse, the American Miniature Horse and the Shetland Pony. To date, no homozygous W13/W13 horses have been identified, suggesting that this could be embryonically lethal.

W5 is found in offspring of the Thoroughbred stallion Puchilingui. **W10** is only found in the offspring of the Quarter Horse stallion GQ Santana. **W22** is found in offspring of the Thoroughbred stallion Airdrie Apache.

W20 has been described in many breeds. This mutation is thought to have a smaller effect on protein function and a more subtle effect on the amount of white expressed, unless it is combined with other dominant white gene variants (and perhaps other chequering patterns). When combined with other gene variants, W20 has been shown to increase the proportion of white patterning, resulting in a pure white or almost completely white phenotype. In contrast to W5, W10 and W22, the homozygous genotype W20/W20 is not lethal.

W22 occurs on the W20 background, i.e. all horses with the W22 mutation also have the W20 mutation. As the W22 mutation has a greater influence on protein function than W20, the allele described is W22, although technically both the W20 and W22 variants are present. In the case where a horse inherits a W20 from one parent and a W20 and W22 from the other parent (which technically means it has two copies of W20 and one copy of W22), it is found to be a compound heterozygous genotype, W20/W22. Horses with this genotype have been shown to have a pure white phenotype.

12.2.10 Dun

Method	TaqMan SNP assay and fragment length polymorphism
Breed	all
Duration	1 – 2 weeks after sample receipt

Phenotype

Dun is a dominant colour dilution gene, which on the one hand changes the base colour of the coat and on the other hand causes so-called "primitive markings". These include the dorsal stripe, zebra stripes on the legs (leg barring) or head (cobwebbing) and shoulder stripes. The dorsal stripe can be seen on all dun horses for life while the other primitive markings may appear additionally.

The effect of the dun gene on the base colours chestnut, bay and black produces a number of colour shades that range from golden to dark grey to olive. Dun is inherited independently of other coat colour genes and can occur in combination with other genes that affect the base colour.

There are 3 alleles that influence the appearance of the dun dilution and the primitive markings: D (dun dilution and primitive markings), nd1 (no dilution, primitive markings may occur in various forms, e.g. the pseudo-dorsal stripe) and nd2 (no dilution, no primitive markings). D is dominant over nd1 and nd2; nd1 is dominant over nd2.

12.2.11 Greying*

Grey horses are carriers of the greying mutation. As the formation of melanoma is directly linked to the greying mutation, it is described together with hereditary diseases (see Chapter 12.1.11. page 93).

12.2.12 Incontinentia Pigmenti (Hyperpigmentation)

Incontinentia pigmenti leads to dermal lesions, it is therefore described together with hereditary diseases as well (see Chapter 12.1.19, page 97.).

12.2.13 Leopard Complex

Method	TaqMan SNP assay
Breed	all
Duration	3 – 5 working days after sample receipt

Phenotype

A single gene inherited in a dominant manner, called leopard complex (LP), is responsible for the development of various white spots and patterns up to "leopard", e.g. in Appaloosa Horses. Depending on the breeding association, the patterns are approved as "few spot leopard", "leopard", "snowcap blanket", "blanket with spots", "varnish roan (marble)", "snowflake", "frosted", "speckled" or "mottled".

Homozygous carriers of the leopard gene (LP/LP) are almost always affected by night blindness (CSNB), whereas heterozygous carriers (LP/lp) are not affected. CSNB is characterised by impaired vision in the dark and is present from birth.

This form of night blindness must be differentiated from CSNB2 in the Tennesssee Walking Horse; CSNB2 is caused by a mutation in a different gene (see section 12.1.3, page 90).

Horses with both the tobiano and the leopard gene are called pintaloosas.

12.2.14 Mushroom

Method	sequencing
Breed	Shetland Pony
Duration	1 – 2 weeks after sample receipt

Phenotype

Mushroom is a dilute coat colour in Shetland Ponies that is characterised by a distinctive sepia-toned body hair colour, often accompanied by a flaxen mane and tail. The genetic variant is inherited in an autosomal recessive mode.

Mushroom dilutes red pigment (phaeomelanin), so it is mostly seen in chestnut coat colour. The mushroom phenotype shows a wide variation of shades and has similarities to cream and silver dilutions, although it is genetically distinct from both.

If the basic colour is bay the mushroom variant leads to a sepia coloured body hair. It has no influence on the black mane and tail, however, because the colour here is caused by eumelanin.

The phenotype of black horses is not modified by the mushroom variant, even if the horse is homozygous.

12.2.15 Pearl

Method sequencing
Breed all
Duration 1 – 2 weeks after sample receipt

Phenotype
In addition to the four most frequent, dominant dilution genes cream, champagne, silver and dun, there is another gene which causes a dilution of the base colour. In Quarter Horses and Paints, this gene was originally called "Barlink factor", but in Spanish horse breeds, such as Andalusians and Lusitanos, it was called "pearl". It is exactly the same mutation, though, and because of the Spanish ancestors of Quarter and Paint Horses, the gene was eventually called "pearl".
In contrast to the other dilution genes, pearl is inherited in an autosomal recessive manner, i.e. only if the gene is homozygous, the base colour of the horse as well as the mane and tail are diluted evenly. A chestnut becomes apricot, a black horse becomes evenly light grey.
Heterozygous presence of the gene does not change the base colour of the horse. Combined with the heterozygous presence of the cream gene (CR/cr and N/Prl), a phenotype is caused which corresponds to homozygous cream (CR/CR). From their appearance, these horses cannot be distinguished from true cremello, perlino or smoky cream.

12.2.16 Roan Zygosity*

Method partner laboratory
Breed breeds on request
Duration 4 – 6 weeks after sample receipt

Phenotype
The roan gene causes a mixture of coloured and white hairs on the body which leads to a "dilution" of the original coat colour. However, there are no "diluted" hairs, but the interspersed white hairs in the coat cause this phenotype. The head, legs, mane and tail remain unaffected by the roan gene and always show the base colour.
Foals are already born with the pattern, though it may not be visible in the foal's coat and only becomes apparent when the coat is changed for the first time.
The white hairs are evenly distributed over the body if the true roan gene is present, not to be confused with the different patterns of white hair called "roaning". Roaning patterns have an uneven distribution of white hairs and the genes responsible for their inheritance have not yet been identified.
Roan is inherited as a dominant gene and can be found in many different horse breeds, e.g. Quarter Horses, Paints, Paso Finos, Paso Peruanos, Welsh Ponies and Belgian Horses, but not in Arab Horses and thoroughbreds.

Although it has been assumed that roan is homozygous lethal, there are reports about Quarter Horses who have passed on 100 % of the gene to their offspring. In these horses, genetic tests have confirmed that the genomic region which contains the roan gene is homozygous.
DNA testing is used to detect mutations which are associated with the roan pattern in Quarter Horses and Paint Horses. The causative mutation has not yet been identified, so the existing test is a marker test.

12.2.17 Sabino 1

Method	sequencing
Breed	all
Duration	1 – 2 weeks after sample receipt

Phenotype
Horses with Sabino 1 show a spotting pattern characterised by white patches of varying sizes and irregular forms and borders. The spots mainly occur on the face, legs and belly. Sometimes, roaning on the belly or all over the body can occur. The spotting pattern can be seen more or less clearly in horses that are heterozygous for the sabino trait, homozygous horses are usually almost completely white from birth. So far, one causative gene could be identified (Sabino 1) but it is likely that there are other genes that are responsible for similar markings.

12.2.18 Silver Dapple

Method	sequencing
Breed	all
Duration	1 – 2 weeks after sample receipt

Phenotype
The silver gene/silver dapple gene is another gene causing a dilution of the base colour. Unlike cream and champagne, it has no influence on phaeomelanin, only the black pigmented areas are diluted. The effect is mainly visible on the mane and tail which are often interspersed with white and grey hairs when the silver gene is present. The gene is inherited in an autosomal dominant manner, i.e. one copy of the gene is sufficient to cause this phenotype.

12.2.19 Snowdrop

Method	sequencing
Breed	Tinker Horse, Gypsy Cob
Duration	1 – 2 weeks after sample receipt

Phenotype
Different gene variants in the SLC45A2 gene are responsible for various coat colour dilutions such as Cream, Pearl or Sunshine. In 2020, a gene variant that caused the brightening of the coat colour could be detected in a Tinker with brightened coat that did not bear any of the known causal gene variants for Cream, Pearl or Sunshine.
This fur colour dilution is called snowdrop and dilutes both red and black pigment, consequently the basic colours chestnut, bay and black are brightened by this gene variant.
The manner of inheritance is autosomal recessive, i.e. only if both alleles of the animal have the variant for snowdrop, the fur colour dilution will occur. To date, the presence of the gene variant in other breeds has not been scientifically described, but cannot be excluded.

12.2.20 Splashed White (SW 1 – 8)

SW 1 – 4

Method	sequencing and fragment length polymorphism
Breed	all
Duration	1 – 2 weeks after sample receipt

SW 5 – 8*

Method	partner laboratory
Breed	all
Duration	4 – 6 weeks after sample receipt

Phenotype
Splashed white is an uneven piebald pattern characterised by an extremely wide blaze or lantern, often in combination with blue eyes and bright white legs. Some, but not all, horses with a splashed white piebald pattern are deaf.
So far, 8 mutations have been identified – SW1, SW2, SW3, SW4, SW5, SW6, SW7 and SW8 – which are responsible for the splashed white markings. The variants are located on the MITF (SW1, SW3, SW5, SW6, SW7 and SP8) and PAX3 (SW2 and SW4) genes.
SW1 has already been detected in many different horse breeds, e.g. Appaloosa, Quarter Horse, Paint Horse, English Thoroughbred, Trakehner, American Miniature Horse, Shetland Pony, Welsh Pony, Icelandic Horse and others.
The **SW2, SW3, SW6** and **SW7** gene variants have so far been identified in the Quarter Horse and the Paint Horse, **SW5** only in the Paint Horse with low frequency.
SW6 was identified as a de novo mutation in a single family. A de novo mutation means that only this individual, its offspring and future generations of horses descended from this animal can have this mutation.
The occurrence of **SW4** has so far only been described in the Appaloosa and the occurrence of **SW8** in the Thoroughbred.
All of these mutations cause a similar phenotype in horses. Splashed white gene variants are inherited as dominant traits with variable expression. This means that one

copy of a SW mutation produces a splashed white phenotype with variable amounts of white. Horses carrying combinations of the splashed white, tobiano or overo lethal white mutations may have extensive white markings or their coats may be completely white.

Homozygous genotypes have not yet been identified for SW3, SW4, SW5, SW6, SW7 and SW8. The occurrence of the gene variant combinations SW1/SW5, SW3/SW5, SW3/SW6, SW5/SW6 has also not yet been detected. This may be due to the fact that the gene variants SW5, SW6, SW7 and SW9 are rare, or that these genotypes are embryonically lethal.

12.2.21 Sunshine

Method	sequencing
Breed	all
Duration	1 – 2 weeks after sample receipt

Phenotype
In addition to the already known colour dilution genes such as Cream and Pearl, a further variant for dilution of the coat colour has been discovered. Sunshine is inherited in an autosomal-recessive manner. This means that only if the gene is homozygous (Sun/Sun) the basic colour of the horse is diluted. It is assumed that this dilution is similar to Pearl. As well as Pearl, Sunshine can interact with Cream to produce pseudo-double Cream dilute phenotypes.

12.2.22 Tiger Eye*

Method	partner laboratory
Breed	Paso Fino
Duration	4 – 6 weeks after sample receipt

Phenotype
Tiger eye is a lightened iris colour that occurs in Puerto Rican Paso Fino horses. In contrast to the brown eyes of most horses, the 'tiger eye' is characterised by a yellow, amber or bright orange colour.
Researchers at the Veterinary Genetics Laboratory investigated the genetic basis of this phenotype and identified two variants in the SLC24A5 gene that are responsible for tiger eye: Tiger Eye 1 (TE1) in exon 2 and Tiger Eye 2 (TE2) in exon 7. The tiger eye phenotype is inherited as a recessive trait. Horses with tiger eye are most frequently homozygous for the TE1 variant (TE1/TE1). Some Tiger Eye horses are compound heterozygous for both variants (TE1/TE2).
Horses with the TE2/TE2 genotype are rare; the one documented case had a very pale yellow/blue iris colour. In contrast to the dilution of iris colour associated with the cream and champagne mutations, there appears to be no correlation between tiger eye and dilute coat pigmentation. The tiger eye phenotype has been observed in all three non-dilute coat base colours (black, reddish brown and chestnut) and in both males and females.

Although TE1 and TE2 have so far only been detected in Paso Finos, it is possible that these variants may explain the lighter eye colour in closely related breeds.

12.2.23 Tobiano

Method	fragment length polymorphism
Breed	all
Duration	1 – 2 weeks after sample receipt

Phenotype
Tobiano is one of the most common pinto patterns in domestic horses. The patches of these horses have smooth edges and distinct borders, the eye colour is usually dark. Tobianos usually have white feet or even more pronounced leg markings, the head markings are comparable to horses without tobiano. The white patches typically cross the dorsal midline in at least one area, but the size of the patch can vary considerably The tobiano pattern is inherited as a dominant trait. Therefore, horses with the heterozygous genotype N/Tob as well as the homozygous genotype Tob/Tob develop the characteristic colour pattern.

12.3 Performance

12.3.1 Predictive Height Test

Method	TaqMan SNP assay, if necessary sequencing
Breed	Warmblood
Duration	3 – 5 working days after sample receipt (1 – 2 weeks for sequencing)

The base exchange of a specific gene locus regulates the expression of the LCORL gene, which apparently influences the growth of the horse. This gene variation affects the withers of warmblood horses. Other factors such as feeding, keeping and rearing of the young horse, but also the mother mare, are essential for the development, though, and therefore influence the size of the horse as well. Horses with the „small" genotype (T/T) that we tested had an average height of 164 cm. Horses with the genotypes C/T and C/C were 4 – 8 cm taller on average. The test does not guarantee an exact stick measure of the horse, but if the genotype is known, the size can be narrowed down. Breeders are thus able to increase the chance of the desired phenotype, if the genotype of both parents is known.

12.3.2 Genetic Test for the Detection of various Myostatin Variants (Speed Gene)*

Method	partner laboratory
Breed	Thoroughbred
Duration	1 – 2 weeks after sample receipt

The protein myostatin is responsible for inhibiting muscle growth. Myostatin and its associated gene MSTN have been detected in humans as well as in different animal species (e.g. cattle, dog, mouse and horse).

Different variants of the myostatin gene are responsible for the development of varying muscle types: "Sprinters", for example, have a very high proportion of muscle mass in relation to their total body weight and are therefore perfectly suited for fast, short races. In contrast, horses which are better suited for long distances are usually lighter, the ratio of muscle mass to body weight is smaller ("long-distance runners").
Horses which are heterozygous for both myostatin variants tend to be most successful over middle distances.
The genetic test for the detection of the different myostatin variants provides information about which racing distance is ideal for the tested horse. However, the test provides no guidance on the actual suitability of the horse as a race horse.

Myostatin – Mutation and Inheritance

The mutation which is responsible for the 2 myostatin variants can be detected by a DNA test.

There are three genotypes:
Genotype C/C (homozygous): The tested horse is homozygous for the C allele of the myostatin gene and could be best suited for short-distance races. Mostly, these horses are very precocious.
Genotype C/T (heterozygous): The tested horse is heterozygous C/T in the myostatin gene. The horse might therefore be best suited for middle distances.
Genotype T/T (homozygous): The tested horse is homozygous for the T allele of the myostatin gene and could be best suited for long distances. These horses tend to be late developers.

12.3.3 SynchroGait (DMRT3)*

Method	partner laboratory
Breed	American Bashkir Curly Horse, American Miniature Horse, American Saddlebred, Appaloosa, Icelandic Horse, Kentucky Mountain Saddle Horse, Mangalarga Marchador, Missouri Fox Trotter, Morgan Horse, Paint Horse, Paso Fino, Paso Peruano, Quarter Horse, Scandinavian Coldblood Trotter, Trotter, Tennessee Walking Horse
Duration	4 – 6 weeks after sample receipt

SynchroGait is a diagnostic DNA test for a autosomal recessive genetic variant (A) that has a major influence on the gait and coordination of horses. The mutation facilitates a lateral foot sequence, which is a basic requirement for the pass, and inhibits the transition from trot or pass to gallop. Variant A has been identified as an important genetic factor for performance in trotting horses and for the ability to race pass in Icelandic Horses.

In Thoroughbreds, AA horses are more frequently used professionally in trotting races; the SynchroGait test is particularly suitable for horses from French Thoroughbred lines. In cold-blooded horses, the presence of the A variant correlates with trotting technique. AA horses have a natural talent for passing and excellent leg coordination when trotting at high speed.
In Icelandic Horses, AA horses have a genetic predisposition to perform five gaits (including tölt and pass). CA and CC horses are more likely to perform only four gaits (tölt, but no pass), whereby CC horses can find it difficult to learn the tölt, especially at the beginning of their training.

12.3.4 Tractability

Method	sequencing
Breed	Thoroughbred
Duration	1 – 2 weeks after sample receipt

The test for tractability is designed to show a horse's willingness to learn or perform. Since the phenotype is only based on statements of the horse owners or people who have contact with the horse, differentiation is difficult. The test does not provide any information concerning the cleverness of the horse. It should also be noted that factors such as anatomical conditions or diseases can strongly influence the tractability, so genotype and phenotype may not be consistent.

12.4 DNA Profiles for Identity and Parentage Testing

Unlike other identification methods, such as microchips or tattoos, DNA profiles cannot be manipulated or destroyed by external factors, e.g. injuries. They remain unchanged for life.
On the one hand, DNA profiles provide a lifelong, doubtless identification of the animal. On the other hand, parentage can be proven with certainty by comparing the genetic fingerprints of the family members.

12.4.1 DNA Profile

Breed	All
Duration	1 – 2 weeks after sample receipt

A DNA profile is based on the testing of highly variable DNA markers (microsatellites), which differ between individuals by their length (length polymorphism). The overall combination of microsatellites results in a DNA profile which is unique for every individual.
In order to create a DNA profile, initially, DNA is isolated from nucleated cells. Then the segments of the DNA to be analysed are multiplied million-fold by PCR.

The length of the microsatellites can be determined by a computer-aided analysis in the "Genetic Analyser". This data is then used to create an individual and reproducible code for each animal.
The DNA profile is unique, with an accuracy of more than 99.9 %. The only exception are monozygotic multiples. For the identification of an animal, its DNA profile is generated and archived in a DNA database. To create a DNA profile, we test the microsatellites markers which are recommended by the "International Society for Animal Genetics (ISAG)". The generated DNA profiles are internationally comparable with laboratories working according to the recommendations of ISAG.

12.4.2 Parentage

The purpose of parentage testing is to clarify whether supposed parents of a certain animal are the true biological parents.
Each offspring receives 50 % of its genetic material from the mother and 50 % from the father. Provided that the maternity is confirmed, all non-maternal markers in the DNA profile of the offspring must have been inherited from the father. If at least two markers in the DNA profile do not match, paternity can almost certainly be excluded. The same applies to the mother.

Similar to DNA profiles, the informational value of parentage testing considerably depends on the number of examined microsatellites. The more highly variable DNA segments are examined in parentage testing, the more precisely false parentage can be detected.

If a pedigree certificate is required, the DNA profile of both the father and mother must be available in addition to the DNA profile of the offspring to be tested. If paternity should be excluded, the DNA profile of all potential fathers should be available in addition to that of the mother. Results are available approx. after about 2 – 3 weeks.

13. Hygiene Examinations

In order to prevent the spread and transmission of infectious agents, good hygiene management in the equine practice/clinic is essential. The quality and efficiency of the hygiene measures should be checked at regular intervals using suitable procedures. Test kits for sampling will be sent to you after ordering the examination.

Steam Sterilizer Control/Heat Sterilizer Control

Routine testing of steam or heat sterilizers is recommended at least every six months. Testing is performed with bioindicators coated with spores of Bacillus atrophaeus (heat sterilizer) or spores of Geobacillus stearothermophilus (steam sterilizer).

Sample material: test kit with bioindicators (+ transport control)

Method: cultural

Procedure: locations in the sterilizer which are expected to have unfavorable sterilization conditions should be selected for bioindicators 1 – 5. Bioindicator 6 (= positive control/transport control (TPK)) is not sterilized. After the sterilization process, all bioindicators should be promptly returned to the laboratory.

Results: 7 working days after sample receipt

Surface Contamination Testing

To evaluate the current level of contamination on surfaces, a surface contamination examination can be carried out by means of contact plates.

Sample material: contact plates

Method: cultural

Procedure: the protruding side of the contact plate is brought into contact with the surface to be sampled for a few seconds with a slight rolling motion and light pressure. Then carefully remove the contact plate from the surface, close it with the corresponding lid and secure it with an adhesive strip. Avoid touching the sides of the culture medium during sampling. Return the sampled contact plates to the laboratory within 24 hours.

Results: 2 – 3 working days after sample receipt

Surface Disinfection Efficacy Testing

To evaluate the efficacy of the surface disinfection process, an examination can be carried out using contact plates. Routine testing is recommended at least every six months.

Sample material:	contact plates
Method:	cultural
Procedure:	the surface to be examined is cleaned, disinfected and allowed to dry completely before sampling. The protruding side of the contact plate is brought into contact with the surface to be sampled for a few seconds with a slight rolling motion and light pressure. Then carefully remove the contact plate from the surface, close it with the corresponding lid and secure it with an adhesive strip. Avoid touching the sides of the culture medium during sampling. Return the sampled contact plates to the laboratory within 24 hours.
Results:	2 – 3 working days after sample receipt

Endoscope Disinfection Control

Test set contains 2 sterile swabs with transport medium and 3 sterile screw cap containers.

Method:	cultural
Procedure:	clean and disinfect the endoscope in the usual manner and then allow to dry. Sample collection should be carried out in pairs and with disposable gloves. Moisten the swabs with sterile 0.9 % NaCl solution and sample the bending section and the distal end of the endoscope with a separate swab each and place the swab in the transport medium. Rinse the instrument channel and the air-water nozzle with 30 ml sterile 0.9 % NaCl solution each and collect the rinsing liquid in separate screw cap containers. Collect a flush sample from the optical rinsing bottle into the third screw cap container. Return all samples to the laboratory within 24 hours.
Results:	3 – 5 working days after sample receipt

Hygiene Inspections on Site and Online

On request, we can carry out hygiene inspections at your practice/clinic. These can be on-site or online with a live stream to our hygiene team in Bad Kissingen. It involves an assessment of the premises of the practice or the vehicles of the mobile practice as well as an assessment of the hygiene management with a follow-up discussion of the results and advice.

Duration: individually between 1 and 4 hours

Preparation of Inspection Report

After the hygiene inspection, we will be happy to prepare your personal hygiene report on request. This includes a summary of the results in tabular form, the documentation of areas in need of improvement with photos as well as recommendations tailored to the results of the inspection.

Duration: individual

Creation of an Individual Hygiene Concept

We offer to create a hygiene concept individually tailored to your practice or help you to improve your existing hygiene concept.

Duration: individual

14. Appendix

14.1 Reference Ranges Horse

Clinical Chemistry

Enzymes 37 °C		
AP	< 352	U/l
AST (GOT)	< 568	U/l
CK	< 452	U/l
GLDH	< 13	U/l
γ-GT	< 44	U/l
α-HBDH	< 221	U/l
LDH	< 455	U/l
Lipase (DGGR)	< 20	U/l

Substrates		
Albumin	25 – 54	g/l
A/G ratio	0.7 – 1.1	
Bile acids	< 12	µmol/l
Bilirubin, total	8.6 – 59.9	µmol/l
Cholesterol	1.81 – 4.66	mmol/l
Creatinine	71 – 159	µmol/l
Fructosamines	< 360	µmol/l
Globulins	24 – 51	g/l
Glucose	3.05 – 4.99	mmol/l
Lactate	0.5 – 2.0	mmol/l
Protein	55 – 75	g/l
SAA	< 7	µg/ml
SDMA	< 0.75	µmol/l
Triglycerides	< 0.97	mmol/l
Urea	3.3 – 6.7	mmol/l

Electrolytes, Trace Minerals and Vitamins		
Calcium	2.5 – 3.4	mmol/l
Chloride	95 – 105	mmol/l
Copper	7.9 – 21.0	µmol/l
Iron	17.9 – 64.5	µmol/l
Magnesium	0.5 – 0.9	mmol/l
Manganese	0.60 – 3.50	µg/l
Phosphate anorganic	0.7 – 1.5	mmol/l
Potassium	2.8 – 4.5	mmol/l
Selenium	100 – 200	µg/l
Sodium	125 – 150	mmol/l
Zinc	5.0 – 14.4	µmol/l
Vitamine E	> 2 1.5 – 2 <1.5	mg/l adequate mg/l marginal mg/l deficient

Urine

γ-GT/creatinine ratio	< 5.0	U/mmol
Protein/creatinine ratio	< 0.5	
Fractional Electrolyte Excretion		
Chloride	0.7 – 2.1	%
Potassium	35 – 80	%
Phosphate	0 – 0.2	%
Sodium	0.04 – 0.52	%

Haematology

Erythrocytes	6 – 12	T/l
Thoroughbred	8 – 12	T/l
Warm blood	6.5 – 9	T/l
Cold blood	6 – 9	T/l
Pony	5.5 – 8.5	T/l

Haematocrit	0.3 – 0.5	l/l
Thoroughbred	0.35 – 0.50	l/l
Warm blood	0.33 – 0.45	l/l
Cold blood	0.32 – 0.42	l/l
Pony	0.30 – 0.40	g/l

Haemoglobin	110 – 170	g/l
Leukocytes	5 – 10	G/l
Platelets	90 – 300	G/l

Differential blood count	%	absolute: G/l
Segmented	45 – 70	3 – 7
Lymphocytes	20 – 45	1.5 – 4
Monocytes	0 – 5	0.04 – 0.4
Eosinophils	0 – 4	0.04 – 0.3
Basophils	0 – 2	0 – 0.15
Unsegmented	0 – 6	0 – 0.6

Coagulation

Thrombin time	18 – 55	sec.
PTT	30 – 65	sec.
Prothrombin time	8 – 14	sec.
Fibrinogen	100 – 350	mg/dl

Endocrinology

Pancreas			
Insulin	<15	µU/ml	negative for insulin dysregulation (ID)
	15 – 35	µU/ml	suspicious for an ID if clinical signs are present
	>35	µU/ml	positive for an ID

Pituary gland / Adrenal cortex					
	PPID	negative	borderline	positive	
ACTH	Dec. – June	< 15	15 – 40	> 40	pg/ml
	July	< 15	15 – 50	> 50	pg/ml
	August	< 20	20 – 75	> 75	pg/ml
	Sept. – Oct.	< 30	30 – 90	> 90	pg/ml
	Nov.	< 15	15 – 40	> 40	pg/ml
Cortisol	Ref. range	30 – 70			ng/ml

Reproduction & Pregnancy				
Oestradiol	reference values mare	pro-oestrus	1.2 – 6.2	pg/ml
		oestrus	7.1 – 13.0	pg/ml
		di-oestrus	3.7 – 5.0	pg/ml
Progesterone			> 1.0 *	ng/ml
Testosterone	reference values	stallion	1 – 5	ng/ml
		gelding	< 0.04	ng/ml
		mare	< 0.04	ng/ml
Anti-Müllerian Hormone		see page 68 and page 70		
PMSG & Oestrone sulfate		depending on stage of pregnancy		

* reflects corpus luteum function

Thyroid gland		
T4	1.3 – 4.1	µg/dl
fT4	9.0 – 44.9	pmol/l
T3	25 – 180	ng/dl

Serum Protein Electrophoresis

Reference range	%	absolute (g/l)
Albumin	45 – 60	33 – 38
α-Globulins	10 – 20	5 – 8
β-Globulins	10 – 25	8 – 14
γ-Globulins	8 – 22	9 – 14

14.2 Reference Ranges Foal

Clinical Chemistry

Age:	Neonate	1 month	2 months	3 months	4 months	5 months	
Enzymes 37 °C							
AP*	3363 ± 1158	1191 ± 336	1166 ± 227	1166 ± 227	1166 ± 227	990 ± 148	U/l
AST (GOT)	311 ± 111	363 ± 43	365 ± 48	356 ± 48	359 ± 32	395 ± 59	U/l
γ-GT	51 ± 47	32 ±14	25 ± 5	25 ± 5	23 ± 5	25 ± 7	U/l
Substrates							
Albumin	30.5 ± 3	29.8 ± 1.4	30.0 ± 1.7	31.4 ± 1.7	32.5 ± 1.9	34.5 ± 1.9	g/l
Protein	54 ± 8	51 ± 4	52 ± 3	53 ± 3	54 ± 3	57 ± 3	g/l
Glucose	7.8 ± 1.1	7.3 ± 0.6	6.6 ± 0.5	6.5 ± 0.5	6.2 ± 0.6	6.0 ± 0.8	mmol/l
Urea*	4.8 ± 3.5	2.8 ± 0.8	3.3 ± 0.7	4.5 ± 0.9	5.9 ± 0.8	5.6 ± 1.3	mmol/l
Triglycerides	0.13 – 0.4	0.16 – 0.48*					mmol/l
Bile acids	21.7 – 81.7	9.0 – 17.1					µmol/l
Electrolytes							
Phosphate anorganic	1.23 – 2.39	1.57 – 3.1				1.54 – 2.46 (6 months)	mmol/l

* AP and urea: higher values in pasture foals
Triglycerides can occasionally be higher in the 1st month of life (0.4 – 2.3 mmol/l)

Parameters that are not significantly influenced by the age of the foals		
Enzymes		
CK	305 ± 150	U/l
Substrates		
Creatinine	89 ± 18	µmol/l
Electrolytes		
Calcium	3.0 ± 0.1	mmol/l
Chloride	102 ± 2	mmol/l
Magnesium	0.7 ± 0.1	mmol/l
Potassium	4.4 ± 0.3	mmol/l
Sodium	138 ± 2	mmol/l

Haematology

Age:	2 days	1 week	1 month	6 months	
Erythrocytes	9.3 – 10.3	8.9 – 9.9	9.3 – 9.9	9.3 – 10.1	T/l
Haematocrit	0.4	0.4	0.3 – 0.4	0.4	l/l
Haemoglobin	133 – 148	123 – 138	114 – 126	119 – 127	g/l
Leukocytes	6.8 – 8.1	9.5 – 10.9	8.4 – 9.8	11 – 12.3	G/l
Platelets	190 – 213	162 – 195	233 – 274	147 – 207	G/l

Differential Blood Count – Absolute Numbers

Age:	2 days	1 week	1 month	6 months	
Segmented	5 – 6.4	6.6 – 7.8	5.1 – 5.9	4.4 – 5.3	G/l
Lymphocytes	1.6 – 1.9	2.3 – 2.7	2.5 – 3.3	5.5 – 6.3	G/l
Monocytes	0.1 – 0.2	0.3 – 0.4	0.3	0.5	G/l
Eosinophils	up to 0.1	up to 0.1	up to 0.1	up to 0.3	G/l
Basophils	0	up to 0.1	0	0	G/l
Unsegmented	– no reference range, pathological –				

Passive Immune Transfer in Neonatal Foals – Serum IgG

< 2 g/l	= absolute deficiency
2 – 4 g/l	= partial deficiency
4 – 8 g/l	= subnormal
> 8 g/l	= adequate supply

14.3 Reference Ranges Donkey

Clinical Chemistry

Enzymes 37 °C		
AP	< 252	U/l
CK	< 525	U/l
AST (GOT)	< 536	U/l
γ-GT	< 70	U/l
GLDH	< 8	U/l
LDH	< 538	U/l

Substrates		
Albumin	22 – 32	g/l
Bile acids	< 18.6	µmol/l
Bilirubin, total	0.1 – 3.7	µmol/l
Cholesterol	1.4 – 2.9	mmo/l
Creatinine	53 – 118	µmol/l
Fructosamines	< 357.6	µmol/l
Globulins	32 – 48	g/l
Glucose	3.9 – 4.7	mmo/l
Protein	58 – 76	g/l
Triglycerides	0.6 – 2.8	mmo/l
Urea	1.5 – 5.2	mmo/l

Electrolytes and Trace Minerals		
Calcium	2.2 – 3.4	mmol/l
Chloride	96 – 106	mmol/l
Copper	9.4 – 18.4	µmol/l
Iron	6.4 – 25.5	µmol/l
Magnesium	0.8 – 1.1	mmol/l
Phosphate anorganic	0.84 – 1.44	mmol/l
Potassium	3.2 – 5.1	mmol/l
Selenium	50.6 – 179.2	µg/l
Sodium	128 – 138	mmol/l
Zinc	3.3 – 14.1	µmol/l

Haematology

Erythrocytes	4.4 – 7.1	T/l
Haematocrit	0.27 – 0.42	l/l
Haemoglobin	89 – 147	g/l
Leukocytes	6.2 – 15	G/l
Platelets	95 – 384	G/l

Differential Blood Count

	%	G/l
Segmented	23 – 59	2.4 – 6.3
Lymphocytes	34 – 69	2.2 – 9.6
Monocytes	0.5 – 7.5	0 – 0.75
Eosinophils	0.9 – 9.1	0.1 – 0.9
Basophils	0 – 0.5	0 – 0.07
Unsegmented	0	0

Endocrinology

Pancreas		
Insulin	< 14.4	µU/ml

Adrenal cortex			
ACTH	Aug. – Oct.	19.5 – 143	pg/ml
	Nov. – July	5.0 – 55.4	pg/ml

Reproduction & Pregnancy				
Testosterone	single determination	donkey stallion	1.24 – 2.11	ng/ml
		cryptorchid	0.32 – 0.58	ng/ml
		gelding	0.01 – 0.09	ng/ml
	HCG Stimulation Test (basal values see above)	cryptorchid (stimulation value)	0.35 – 0.75	ng/ml
		gelding (stimulation value)	0.01 – 0.12	ng/ml
AMH	reference value	male, completely neutered	< 14.7 ± 2.4	ng/ml

Thyroid gland		
fT4	5.0 – 6.2	pmol/l
T4	2.59 – 4.10	µg/dl
T3	51.9 – 77.7	ng/dl

In younger donkeys (< 5 years), elevated thyroid values can sometimes be measured, in older donkeys (> 11 years) lower values.

15. Finally, a few Words on the Process

15.1 Courier Service

LABOKLIN offers courier services in most EU-countries. The samples are generally delivered to LABOKLIN within 24/48 hours. For more information, including prices and the possibilities of sample collection in your area, please contact our Service Department or your local LABOKLIN office.
Our contacts: See page 8 and following.

15.2 Invoicing

All prices listed on the submission forms are quoted without the applicable Value Added Tax (VAT). To receive VAT-free invoices, please provide your international tax number (EU only). We issue invoices at the beginning of the next month with detailed information on costs per sample and tests performed in the previous month, together with animal and owner name. If an invoice is to be sent to the owner, we invoice with a factor 1.4 plus 19 % German VAT. This is only possible for genetic tests, and when the owner's signature and complete data are supplied.
There are discounts available to veterinarians depending on the monthly invoice revenue: For more information, please contact us or your local LABOKLIN office.

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Notes

LABOKLIN
LABORATORY FOR CLINICAL DIAGNOSTICS

Luteo-placental Shift & Placental Function

Two important criteria for assessing pregnancy in horses

Image source: envatoelements

Exclusively at Laboklin

Why determine the luteo-placental shift?

Knowing the timepoint of the luteo-placental shift (DHP/progesterone ratio) helps in deciding the appropriate period for progestagen administration during pregnancy in problem mares.

What else can DHP (5α-dihydroprogesterone) tell you?

Absolute values (follow-up examinations) may help you access the placental function in the case of disease during advanced pregnancy.

Questions?
Our equine vets will be happy to guide you!

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