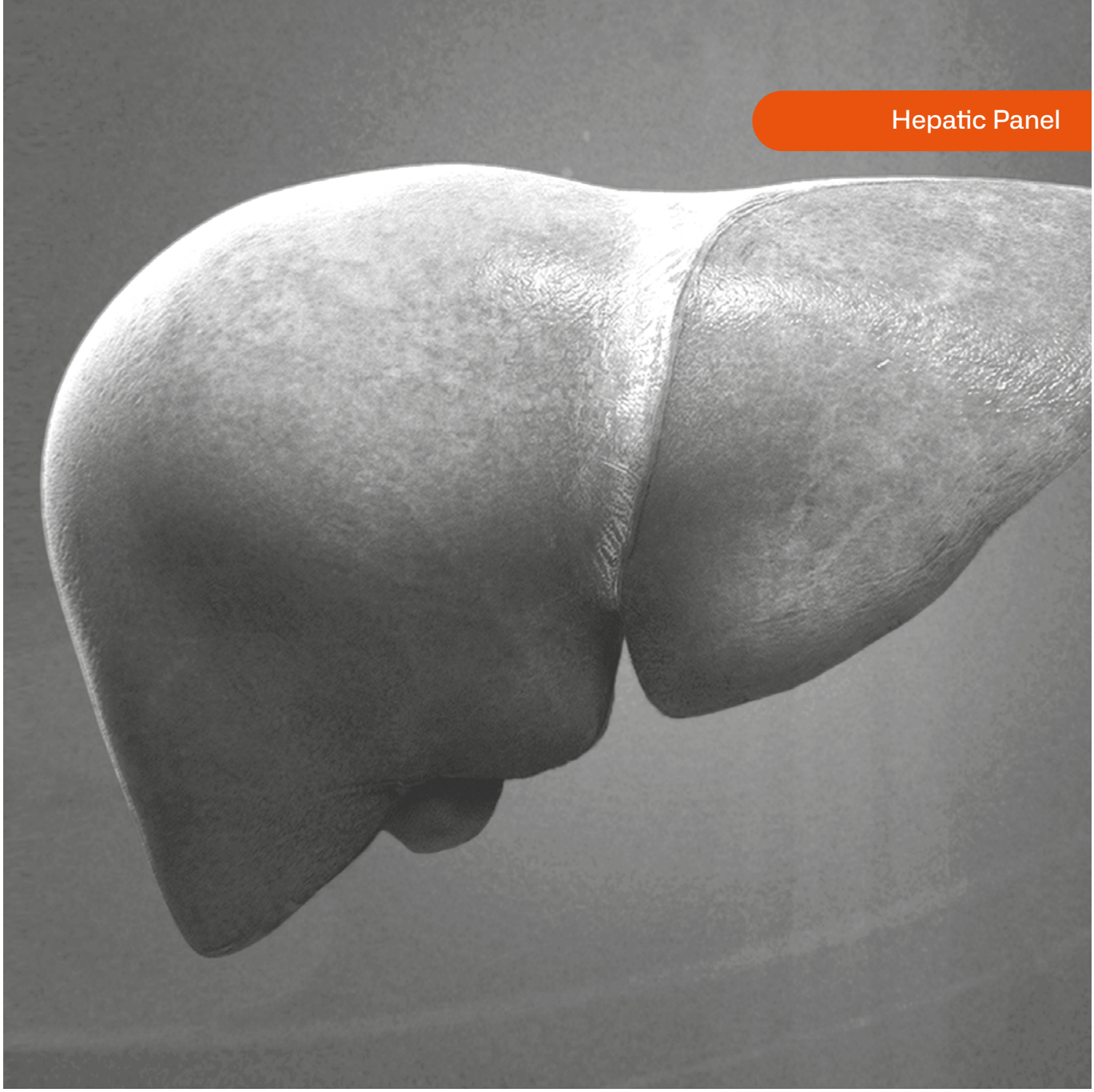


# Product Sheet
# **Ammonia**

BioSystems

Clinical analysis

human - centred biotech

# Ammonia

Hepatic Panel

## What is ammonia?

**Ammonia** is a waste product of the protein catabolism and other nitrogenous compounds. It is formed mainly in the intestine by the degradation of proteins and other nitrogenous compounds, in the purine nucleotide cycle and in amino acid transamination cycles in skeletal muscle and by other metabolic processes in kidneys and liver. Ammonia is metabolized to urea in the liver by different enzymatic reactions and is eliminated from the body through the urine.

## Why measure ammonia?

Reagents for the measurement of ammonia concentration in plasma for the assessment of its imbalance in the general population. Several diseases, acquired and inherited, that involve ammonia elimination are the causes of the hyperammonemia. In pediatrics, increases in ammonia concentration are associated with inborn errors of metabolism related to the urea cycle. Hyperammonemia acquired diseases are caused by severe liver damage, Reye's syndrome, organic acidemia and some kidney diseases. Furthermore, ammonia is a highly neurotoxic compound which is involved in the development of hepatic encephalopathies.

## Reference values and pathologies

**Normal values:**
- Newborns: 90-150 µgN/dL (64-107 µmolN/L)
- Adults: 15-45 µgN/dL (11-32 µmolN/L)

These ranges are given for orientation only; each laboratory should establish its own reference ranges

**Decreased levels:**
- Some types of hypertension
- With the use of some antibiotics, such as neomycin

**Increased levels:**
- Acute or chronic liver encephalopathy, caused by the accumulation of ammonia in the brain due to a decrease in liver function (cirrhosis or hepatitis)
- Decreased blood flow to the liver
- Severe liver damage
- Organic acidemia
- Gastrointestinal bleeding
- With the use of turnstiles
- Enzyme disease of the urea cycle
- Some kidney diseases such as kidney failure
- Some drugs and smokes
- In neonates: Reye's syndrome, severe liver damage, congenital errors of metabolism related to the urea cycle and Hemolytic disease of the newborn

## Method


## Performance characteristics

| | |
|---|---|
| **Method:** | Glutamate Dehydrogenase |
| **Analysis mode:** | Diferential bireagent |
| **Detection limit:** | 26.2 µmol/L = 44.5 µg/dL |
| **Linearity limit:** | 600 µmol/L = 1022 µg/dL |
| **Wavelength:** | 340 nm |
| **On board stability:** | 40 days |
| **Repeatability:** | 1.4% at 297 µmol/L = 505 µg/dL |
| **Reproducibility:** | 2.6% at 297 µmol/L = 505 µg/dL |
| **Sample type:** | Plasma collected using standard procedures |
| **Interferences:** | Bilirubin (up to 30 mg/dL), hemolysis (hemoglobin up to 50 mg/dL) do not interfere. Lipemia and turbidity in the sample interfere with ammonia measurement. Other drugs and substances may interfere |

## Reagents

| Product | Code | Kit Format | Format |
|---|---|---|---|
| **A15/A25 Automated System** | 12532 | 1 x 20 mL + 1 x 7 mL | Lyophilized RA; Liquid RB |
| **BA Automated System** | 23532 | 1 x 20 mL + 1 x 7 mL | Lyophilized RA; Liquid RB |
| **Ammonia, Ethanol, CO2 Calibrator** | 18065 | 2 x 5 mL | Liquid |
| **Ammonia, Ethanol, CO2 Control level I** | 18063 | 3 x 5 mL | Liquid |
| **Ammonia, Ethanol, CO2 Control level II** | 18064 | 3 x 5 mL | Liquid |

customersupport@biosystems.es

**BioSystems S.A.**
Costa Brava 30, 08030 Barcelona (Spain)
t. +34 933 110 000
www.biosystems.global

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