HEALTHWARE

Actualities of the Hungarian pharmaceutical financing market

Newsletter
No. 3, Issue VI. 2018
Published: 21/03/2018

News, current issues

News Healing is running mainly withouth guidelines »

News Tamás Freund: Alzheimer's disease may become curable »

News Sheeppox vacination will be free of charge from next January »

Decision-making index, January 2018

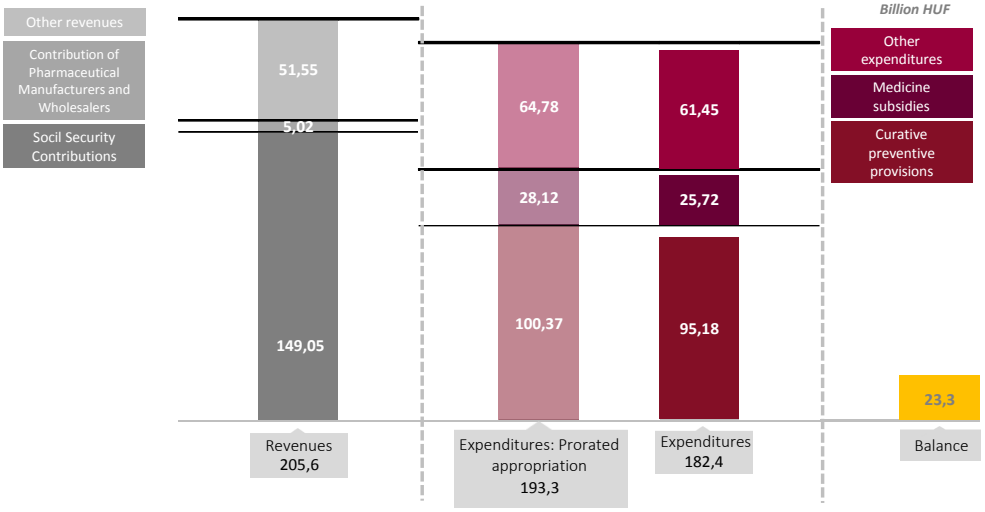
2,42

5/257	Legislation
130/257	Activity of Parliament
9/257	NIHIFM decisions

Methodology

Macro approach to financing healthcare and medicinal products

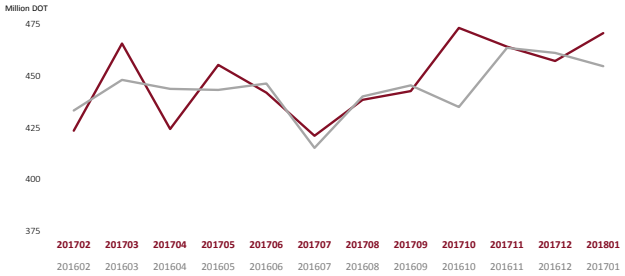
Balance of the Health Insurance Fund, January 2018



Source: Healthware analysis based on NHIFA data

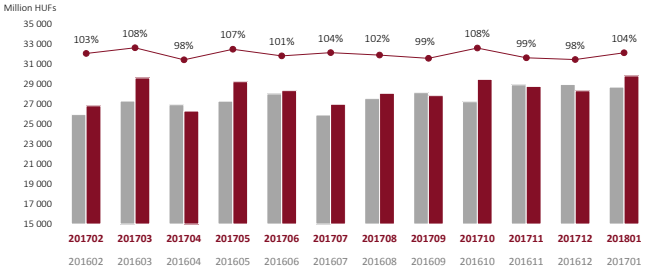
Dynamics of the sales/circulation of prescription-only-medicine

Pharmacy DOT turnover



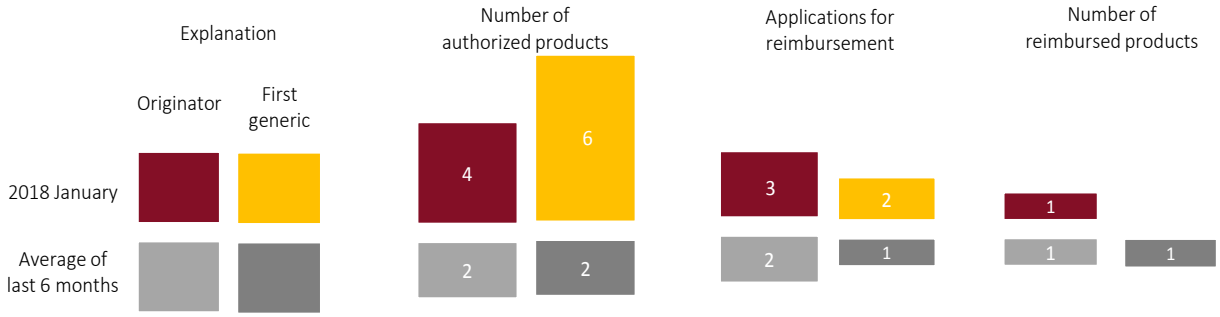
Source: Healthware analysis based on NHIFA data

Pharmacy reimbursement turnover



Source: Healthware analysis based on NHIFA data

Changes to subsidized medicinal product categories, January 2018



Source: Healthware analysis based on NHIFA data

Product offering

FX-process/Reference pricing

Following the changes eventuated in the course of formation of FX-groups:

- Presentation of changes in group-and product level
- Modelling of forming of FX-groups:
  - Cancel and create groups,
  - Combine and dissociate groups,
  - Cancellation of products,
  - Translocation of products,
  - Change of price, reimbursement and DOT-values of products
- Analysis related to FX-process

According to the demand of Client we make decision preparatory and modelling analysis about fix groups related to the portfolio of our Partner.

Further information about the service: **link**

Healthware Consulting Ltd.
H-1093 Budapest Közraktár st. 30-32. 7th floor. I + 36-1-324-2050

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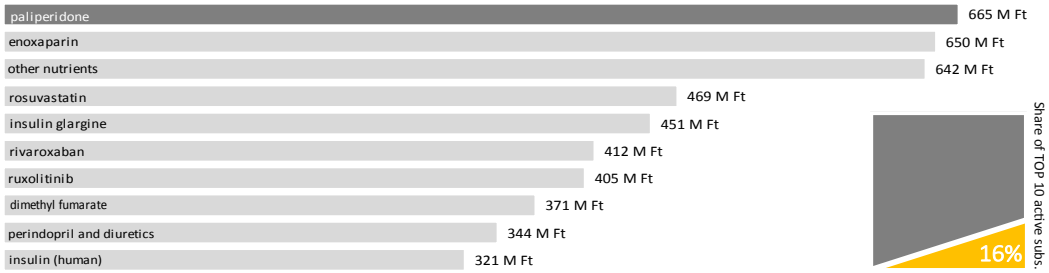
Market data

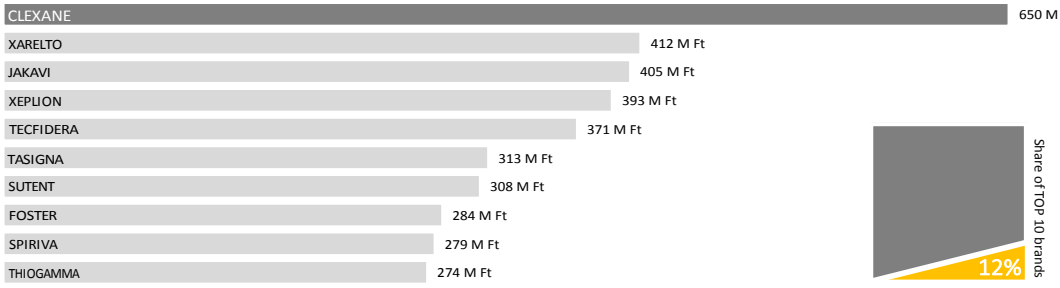
Toplists of reimbursement and number of patients, January 2018

TOP 10 ATCs by all reimbursement paid



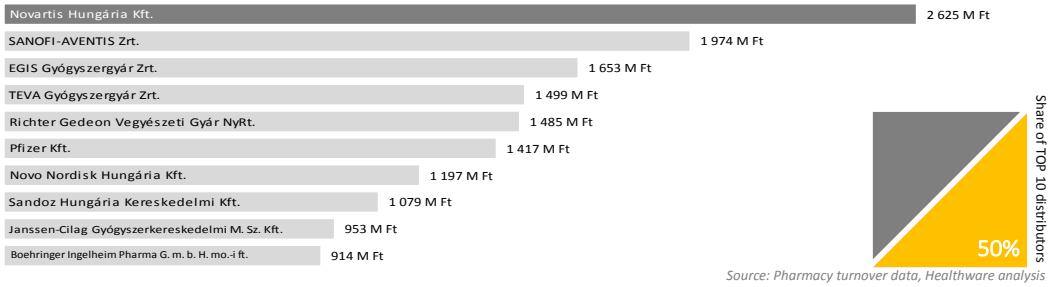
Source: Pharmacy turnover data, Healthware analysis

TOP 10 brands by all reimbursement paid



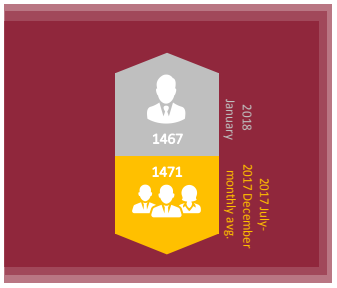
Source: Pharmacy turnover data, Healthware analysis

TOP 10 distributors by all reimbursement paid



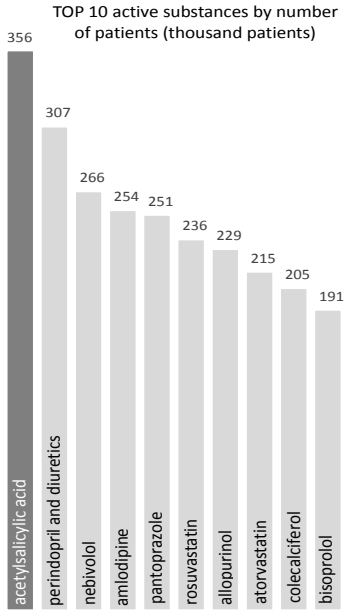
Source: Pharmacy turnover data, Healthware analysis

Average number of medical sales reps



Source: NHIFA data, Healthware analysis

TOP 10 active substances by number of patients (thousand patients)



Source: Pharmacy turnover data, Healthware analysis

Mendelian randomization – Case study

Understanding of the relationship between a particular disease and its potential risk factors is important in preventing or early detection of a disease. These relationships can take several forms:

1. The risk of the development of the disease is increased by the risk factor (causal relationship).
2. The existence of the apparent risk factor is the consequence of the disease itself (reverse causation).
3. The true cause of the disease is also related to the apparent risk factor (confounder).

Randomized controlled trials are the classical and up to now most effective means to investigate causal relationships. Treatment and control groups are constructed by randomization and later the groups are exposed to different levels of the risk factor. If significant differences of the incidences and severities of symptoms of the disease are observed between groups then they could only be caused by the risk factor (or eventually a freak of chance).

Randomized controlled trials are not always feasible due to ethical, financial reasons, or time limitations. In certain cases however, observational data can be used to explore causal connections if randomization into groups is substituted with the random genetic assortment processes of conception. This method is referred to as Mendelian randomization. Its conditions of applicability are as follows:

1. It is possible to identify a locus on the chromosome where the levels of the risk factor differ between the genotypes at that locus. The groups of the corresponding Mendelian randomization are patient members of these genotypes.
2. The genotypes are not related to any environmental, social or demographic confounding factors. (It can usually be presumed, as genotype is determined at the moment of conception. However, it is still worth checking statistically.)

When the conditions are met and significant relationship is detected between genotypes and disease incidence or severity then this demonstrates the causal effect of the risk factor. Consequently, Mendelian randomization is suitable to investigate causal connections. A detailed review of the method providing a number of illustrating examples is (Smith & Ebrahim 2003).

We illustrate the structure and effectiveness of Mendelian randomization by a computer simulation experiment. Let the genotypes be G1, G2, G3. We suppose that the theoretical proportions of genotypes among patients are the same (each of them is equal to 33.3%). The risk factor is generated as a normally distributed random variable in each genotype group with the following parameters:

	Mean	*SD*
G1	6	2
G2	7	2
G3	8	2

Disease prevalence follows a logistic regression model depending on the risk factor, according to the following formula:

log(prevalence/(1-prevalence)) = -9+risk factor.

We simulated the data of 1000 patients. The following descriptive statistics were obtained:

	Number of patients	*Mean (SD) of the risk factor*	*Disease prevalence (%)*
G1	363	5,9 (2,0)	13,8
G2	308	7,0 (1,9)	21,4
G3	329	8,2 (2,1)	37,1

The summary table shows that – as a causal consequence of the risk factor – disease prevalence differ relevantly by genotypes. It can be demonstrated by fitting logistic regression model that the differences are significant at 5% significance level. This underpins the existence of the causal relationship between disease prevalence and the risk factor.

Based on these it can also be stated that the genotype examination has a major role in understanding the relationship between diseases and a possible risk factor, as by using Mendelic randomization, a similar conclusion (cause and effect connection verification) can be concluded, like at randomized controlled experiments.

Reference:

Smith GD, Ebrahim S (2003). 'Mendelian randomization': can genetic epidemiology contribute to understanding environmental determinants of disease? International journal of epidemiology 32: 1-22.