# NeuroFAST Roadmap

At our final meeting, we reflected over current (2015) key gaps in knowledge with a view to the provision of a roadmap beyond this project for future EC research.

| OUTCOME | FUTURE |
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| We provide evidence that subtypes of obesity exist and show that neither being “Food/Eating addicted” nor being “stressed” are consistent determinants of becoming overweight or obese. | If we are to develop personalised strategies for overweight and obesity, future research will need to address why different people respond differently in terms of weight gain to the same environmental stimuli that include stress. |
| We provide a platform for a working definition of the term “Food Addiction” (or “Eating Addiction”, as we prefer to describe it). We made progress towards its validation in obese women by brain imaging and also show that being food addicted is not a constant determinant of being overweight/obese. | Further validation and implementation of clinical tools for the diagnosis of “Eating addiction” is warranted – this evidence base is needed to structure policies that guide the environment of those affected and also that provide better health care for those diagnosed. |
| We show that the reward value of food and anticipatory behaviours for it are not only associated with its taste but are also influenced by our gut, exemplified here by the effects of ghrelin on reward behaviour for food. | Appetite-regulating hormones provide a window on brain function for appetite control: by studying their brain mechanism at many levels, we can discover new mechanisms for appetite control – both how much and what we eat. |
| We show that diet impacts on certain food-linked behaviours that can be obesity-promoting but find little evidence to support the idea that the pathways engaged by foods are those that orchestrate addiction-like behaviour. | We need to strengthen behavioural science showing impacts of diet on subsequent choice behaviour for food and the environmental influences involved. |
| Research on causal factors underlying diet induced alterations in energy balance showed that energy-sensors such as AMPK or mTOR are at the core of orchestrating a variety of brain functions (body temperature, energy and metabolic homeostasis) in response to nutritional status and/or diet intake. | As behavioral models in rodents become more sophisticated and can be validated to some extent in a non invasive way in humans it should be feasible to carry out the circuitry analysis linking energy sensors and neuropeptides/ neurotransmitters involved in diet-induced alterations in energy and metabolic homeostasis opening the way for novel clinical approaches to treat obesity and comorbidities. |

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