Topic: Development and validation of translational platforms in support of synaptopathy drug discovery

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Topic details

Action type Research and Innovation Action (RIA)
Submission and evaluation process 2 stages

Specific challenges to be addressed

Central nervous system (CNS) disorders are some of the most prevalent, devastating and poorly treated illnesses that impact individuals, families and society. It is estimated that annually in Europe alone, approximately 38% of the population will suffer from a CNS disorder. When adjusted for age and comorbidities, this equated to 164.8 million people in 2010 and notwithstanding the emotional and social burden to patients and families, the financial cost was determined to be approximately €798 billion [1]. The brain due to its unique complexity is susceptible to a variety of CNS disorders that can develop throughout all phases of life. For example, neurodevelopmental illnesses such as autism spectrum disorders first appear in early childhood, whilst psychiatric disorders are typically diagnosed during teenage years or early adulthood, and as we age we become increasingly susceptible to Alzheimer’s disease, Parkinson’s disease and other neurodegenerative diseases. As a direct consequence of disease complexity and heterogeneity, identifying commonalities between CNS disorders has proved somewhat elusive, but there is now mounting evidence demonstrating that subtle, albeit persistent disturbances in synaptic functioning may underlie a number of brain disorders.

The term ‘synaptopathy’ first coined in 2003 is typically used to describe CNS disorders caused by synaptic deficits, irrespective of whether the alterations are primary or caused by underlying pathophysiological processes [2]. Synaptic deficits can be triggered by changes in the intrinsic pre- or post-synaptic molecular machinery or by alterations in the surrounding synaptic environment. For example, approximately 600 genetic variations, many of which specifically affect synaptic proteins are linked to autism spectrum disorders and despite the considerable diversity of these genes, many of these map onto common synaptic pathways [3]. Impaired synaptic function is also a core feature of several neurological disorders including Alzheimer (AD) and Parkinson disease (PD). AD brains contain extracellular deposits of amyloid beta (Aß) peptide and intracellular neurofibrillary tangles (NFTs), mostly composed of aggregated tau protein. Although several hypotheses have been proposed for the cause of AD, the most common being the amyloid cascade hypothesis [4][5] and the tau hypothesis [6], the precise mechanisms of Aß and tau toxicity are still not fully understood. It is noteworthy that neither plaques nor NFT volumes correlate well with disease severity, whereas the loss of nerve endings and associated synaptic dysfunction more closely track cognitive impairments [7][8][9].

Within psychiatric disorders, several studies have demonstrated that major depressive disorder is associated with profound reductions in key brain regions that regulate mood and cognition, including the prefrontal cortex and the hippocampus and that these areas show significantly reduced synapse numbers. Antidepressants have been shown to block or reverse these deficits. In addition, and more recently, ketamine, an N-methyl-D-aspartate receptor antagonist that elicits a rapid antidepressant response in treatment resistant patients has been shown to elicit synaptogenesis and reverses synaptic deficits caused by chronic stress in preclinical species [10]. Finally, one of the most consistent observations from schizophrenia patients is pronounced grey matter loss, which is accelerated during adolescence. Several post-mortem studies have demonstrated spine density alterations in the
brain regions showing the greatest grey matter loss and these results support the notion that spine density changes directly contribute to grey matter loss [11].

Whilst our emerging understanding of how synapses are pathologically altered in certain brain disorders is leading to innovative opportunities for drug discovery, there are considerable challenges impeding effective research that still remain. For instance, whilst there has been some recent headway, many of the utilised preclinical disease models, both in-vitro and in-vivo are typically selected based on tenuous links to alterations in synaptic pathology. This is a direct consequence of the fact that many of the disease, pharmacodynamic and efficacy models were developed and validated on historical neurotransmitter modulation approaches and whilst successful in their day, may not prove to be amenable for synaptopathy drug discovery. Furthermore, the current technologies and platforms employed within early drug discovery are not fully characterised with respect to their predicative translational value thus leading to a high risk of failure once compounds are progressed into the clinic. What is desperately needed therefore is the identification and validation of robust, sensitive and translational platforms capable of quantifying synaptic alterations both preclinically and clinically. Such platforms should be fit for purpose to detect and quantify dynamically both disease and treatment effects. Finally, we need to demonstrate the value of these new tools and methods for supporting drug discovery and development efforts across a spectrum of therapeutic CNS indications, including neurodegenerative, neurodevelopmental and psychiatric disorders. This will allow showing synaptopathy as a fundamental treatable trait of these otherwise diverse conditions and will foster a leap forward towards innovative medicines for these diseases.

**Need and opportunity for public-private collaborative research**

CNS disorders are a ticking time bomb under the European economy due to the considerable societal costs and to the fact that these expenses will increase exponentially due to an ever-growing aging population. Despite this concern and the current efforts of the European scientific community, there is still a major discrepancy between the impact of CNS disorders and the modest resources that are directed to brain research. At the other end of the value chain, innovative treatments for patients are lacking. To improve efficiencies and ultimately drive success, it is imperative that intensive, collaborative research programmes are implemented. These should connect experts across sectors and disciplines, breaking silos and allowing pooling of resources and expertise from industry, academia and Small and Medium-sized Enterprises (SMEs). Only such partnership can ultimately deliver a heightened understanding of the contribution of synapse dysfunction to CNS disorders together with a battery of robust, validated, decision-making preclinical and clinical platforms to facilitate drug development. Expertise in drug discovery and development from industry, and academic expertise ranging from basic to clinical neuroscience should be brought together. Integration of SMEs which can play an important role as innovators in the field is also critical. Finally, yet importantly, patients and regulators must be part of the collaborative research efforts to ensure significant impact. The Innovative Medicines Initiative public-private partnership model is best placed to implement such collaborations to achieve a leap in scientific understanding and deliver a robust and highly validated platform of tools and technologies that can be exploited to deliver much needed novel CNS medicines.

**Scope**

The science linking alterations in synaptic function, genetics, and underlying pathways with CNS disorders is emerging. What still needs to be addressed is how these alterations are causal in the development of brain disorders, if they represent a common pathophysiological mechanism across disorders, and, finally, if targeting such alterations is feasible for the development of new treatments. The overarching aim of this topic is to develop an improved understanding of the causative or contributory role of synaptic alterations in CNS disorders, which must be valid and applicable to drug discovery and development across the diverse therapeutic CNS areas. The aim is to construct a precompetitive research consortium focussed on furthering our scientific understanding on how synaptopathies can elicit or contribute to brain disorders. In addition, the focus will be to develop and validate both existing and innovative translational tools and platforms to facilitate drug discovery targeting synaptic health. If successful, the knowledge and validated technologies derived from this effort will facilitate the delivery of promising pharmaceutical therapies for the treatment of CNS disorders, for example neurodevelopmental, psychiatric and neurodegenerative disorders that are linked to deficits in synapse function.

To achieve the overall aim of the topic, applicants should focus on at least one of the four major brain disorders namely Alzheimer’s, Parkinson’s disease, major depression and schizophrenia, and ideally at least two, one in the
neurodegenerative and one in the psychiatric/neurodevelopmental field. In their short proposal the applicants should convincingly address how their approach and specifically their choice of technologies, disease models, preclinical and clinical platforms together with selected patient cohorts is optimal for achieving the topic objectives as outlined below:

1. Bolstering scientific understanding on how synaptic alterations cause or contribute to CNS disorders and pave the way to efficient and effective synaptopathy drug discovery, with demonstration of the applicability beyond an individual brain disorder and its specific pathophysiology;
2. Developing and characterising of in-vivo preclinical models of synapse function using both existing and innovative technologies to identify those that demonstrate improved sensitivity and predictive translational value;
3. Developing and characterising of both existing and novel clinically applicable platforms and treatment sensitive biomarkers capable of quantifying synaptic health leading to the selection of improved endpoints for use in patient studies.

Specifically, the effort should be divided into two key areas:

**1. Deep clinical phenotyping of CNS disorder patients to enable the development of robust tools to measure disease and treatment effects on the synapse**

Although the science concerning synapse physiology and function and its contribution to brain disorders is emerging, systematic clinical phenotyping of CNS patient cohorts using platforms/technologies including but not exclusive to imaging, electrophysiology and clinical assessment scales are required to strengthen the fundamental knowledge base and identify clinical measurements with heightened sensitivity for disease and treatment effects. To this end the most appropriate patient populations (including at least one of the four major brain disorders namely Alzheimer’s, Parkinson’s, major depression and schizophrenia) and assessment platforms should be selected and utilised to deeply phenotype CNS patient cohorts. This should allow the delivery of robust platforms/technologies for clinical measurement of disease and treatment effects on the synapse, and a significant leap forward in the knowledge base of synaptopathy in the context of major brain disorders.

**2. Characterisation of existing and development of novel preclinical synaptopathy disease models**

Although a variety of in-vivo disease models are available for CNS disorder research, the robustness of the reported phenotypes and their translational value in supporting drug discovery efforts requires strengthening. Thus, multiple cross-site characterisation of disease models utilising both available and innovative technologies are necessary to better define and select those most appropriate for drug discovery and development efforts with a focus on the synapse. Disease models may include, but are not limited to, transgenic mouse strains expressing risk genes for psychiatric, neurodegenerative and potentially developmental disorders and also of perturbations known to impact synaptic remodelling.

The applicants should demonstrate their strategy for the choice of the most appropriate models and tools for achieving the objectives of the topic. Technologies and platforms may include synaptic imaging markers as well as other functional correlates such as electrophysiological (Multiple Electrodes Arrays (MEA), Long Term potentiation/Long Term Depression (LTP/LTD), electroencephalograms (EEG), Event Related Potentials (ERPs)), imaging (calcium, high content, immunohistochemical, autoradiography, 2-Deoxyglucose (2-DG)), synaptic biomarkers measurements (Synaptosomal-associated protein 25 (SNAP-25), Growth Associated Protein 43 (GAP-43) etc), microdialysis, neurotransmitter sensors and optogenetics and behavioural platforms (cognitive, motor and psychosis/mood related).

**Expected key deliverables**

**Initial phase (approx. 3 years)**

1. A prioritised list of robust disease models, preclinical and clinical platforms fit for purpose for synaptopathy drug discovery;
2. In-vivo synaptopathy disease models that have been characterised and validated cross-sites using the predefined platforms and technologies to identify those disease models and platforms most optimal for drug discovery efforts;

3. A robust clinical assessment battery able to detect synaptic alterations in relevant patient cohorts;

4. Selected CNS disorder animal models that have been both behaviourally and deeply phenotyped to establish the translation between synaptic marker and behavioural endpoints;

5. Initial interactions with patient groups and regulatory bodies to discuss appropriate development paths forward for novel therapies targeting synaptopathies.

Late phase (approx. 2 years)

Based on successful achievement of the above deliverables, the remaining two years should deliver:

1. A comprehensive cross-site profiling of existing and novel therapies believed to positively address synaptopathy in the defined in-vivo disease models using the battery of preclinical platforms;

2. A definitive clinical evaluation of novel Positron-emission tomography (PET) ligands targeting pre- and post-synaptic proteins, for example synaptic density protein 95 (PSD-95), vesicular glutamate transporters (VGLUT1/2), excitatory amino acid transporter-2 (EAAT2) or others to be determined;

3. The determination of the pharmacological sensitivity of the defined clinical assessment battery using existing chemical entities thought to modulate synaptic function, for example ketamine or others to be determined;

4. Some conclusions, based on the discussions of the results achieved with regulatory bodies and patients, on best development paths forward for novel therapies targeting synaptopathies.

Expected impact

The overarching objectives of IMI2 JU are to develop the next generation vaccines, medicines and treatments to provide patients, with more efficient and effective therapies. The IMI2 JU strategic research agenda (SRA) identified four key focus areas where multi-stakeholder collaborative efforts were deemed critical for success. This topic not only aligns with two of the four strategic research agenda areas (target validation/biomarker research and innovative medicines), but also aims to address three of the twelve IMI2 health priority disorders (neurodegenerative, psychiatric and age-associated diseases).

The expanded knowledge base generated to define the contribution that synaptopathies play in neurodevelopmental, psychiatric and neurodegenerative disorders will lead to improved disease pathway understanding and thus better position academia, SMEs and pharmaceutical companies to identify and validate tractable drug targets. The concerted and aligned efforts will minimise duplication and redundancy. The tools, platforms and technologies will ultimately drive success in both the discovery and clinical arenas by providing robust translatable evidence of early clinical efficacy as compounds are evaluated in patient populations. These achievements will facilitate the delivery of much needed, highly effective medicines and treatments for CNS disorders.

Applicants should also indicate how they will strengthen the competitiveness and industrial leadership of Europe by, for example, engaging suitable SMEs. Solutions that are co-created with SMEs can provide an economic stimulus that can be enduring. Their involvement in the action might offer a complementary perspective to industry and the academia, and help deliver the long-term impact of the project.

Potential synergies with existing consortia

Applicants should take into consideration, while preparing their short proposal, relevant national, European (both research projects as well as research infrastructure initiatives), and non-European initiatives. Synergies and complementarities should be considered to incorporate past achievements, available data and lessons learnt where possible, thus avoiding unnecessary overlap and duplication of efforts and funding.
Potential consortia synergies include:

- IMI-PRISM: https://prism-project.eu/en/prism-study/
- IMI-EU AIMS: https://www.eu-aims.eu/
- IMI-European Prevention of AD: http://ep-ad.org/
- European Lead Factory: https://www.europeanleadfactory.eu/

Industry Consortium

The industry consortium (EFPIA and Associated Partner) will contribute the following expertise and assets:

Preclinical

1. In-vivo disease models known to demonstrate synaptic dysfunction linked to neurodevelopmental, psychiatric and neurodegenerative disorders including but not exclusive to:
   - Transgenic mouse strains expressing risk genes for neurodevelopmental, psychiatric and neurodegenerative disorders;
   - In-vivo viral transduction models;
   - In-vivo proteinopathy seeding and spreading models.

2. Access to technologies, know-how and protocols including but not limited to:
   - Rodent PET;
   - Electrophysiological (MEA, LTD/LTP, EEG, ERP’s);
   - Imaging (calcium, high content, immunohistochemical, autoradiography, 2-DG);
   - Synaptic fluid biomarkers measurements (SNAP-25, GAP-43, Lysosome-associated membrane protein 2 (LAMP-2) for example);
   - Microdialysis, neurotransmitter sensors and optogenetics;
   - Behavioural platforms (cognitive, motor and psychosis/mood related).

3. Commercially available and development tool compounds
   - PET ligands for synaptic markers;
   - Pharmacological modulators of synaptic architecture.

Clinical

1. Clinic ready PET ligands (SV2A and AMPA TARP) in addition to PET chemistry support for novel ligand development;
2. Clinical expertise in trial design, implementation and regulatory support.
The industry consortium may also support communication/dissemination and project management activities.

**Indicative duration of the action**

The indicative duration of the action is 60 months.

**Applicant Consortium**

The applicant consortium will be selected on the basis of the submitted short proposals. The applicant consortium is expected to address all the research objectives (please refer carefully in particular the sections “Scope”, “Deliverables” and “Suggested architecture of the full proposal”) and make key contributions to the defined deliverables in synergy with the industry consortium, which will join the selected applicant consortium in preparation of the full proposal for stage 2. This may require mobilising, as appropriate, the following expertise:

- Basic neuroscientists with disease understanding of neurodevelopmental, psychiatric and neurodegenerative disorders;
- Clinical and disease area experts with access to patient cohorts;
- PET ligand development experts;
- Imaging, electrophysiology and fluid biomarker experts;
- Expertise in clinical data management and clinical statistics;
- Expertise in patient advocacy and engagement, privacy and ethical considerations;
- Regulatory expertise and experience in development and qualification of novel end-points.

The participation of SMEs with the following expertise is highly encouraged:

- PET ligand development;
- Imaging and image analysis technologies;
- Clinical trial operation and execution;
- Targeted mass spectrometry based proteome analysis;
- Data and knowledge management;
- Project management with expertise and experience relevant to IMI2 JU/H2020 projects.

Addressing successfully the objectives of the topic may also require mobilising, as appropriate, the following resources (please refer carefully in particular the sections “Scope”, “Deliverables” and “Suggested architecture of the full proposal”):

- Patient cohorts;
- Patient and regulatory bodies.

**Suggested architecture of the full proposal**

The applicant consortium should submit a short proposal which includes their suggestions for creating a full proposal architecture, taking into consideration the industry contributions and expertise provided below.

The final architecture of the full proposal will be defined by the participants in compliance with the IMI2 rules and with a view to the achievement of the project objectives.

In the spirit of the partnership, and to reflect how IMI2 Call topics are built on identified scientific priorities agreed together with EFPIA beneficiaries/large industrial beneficiaries, it is envisaged that IMI2 proposals and projects may
allocate a leading role within the consortium to an EFPIA beneficiary/large industrial beneficiary. Within an applicant consortium discussing the full proposal to be submitted at stage 2, it is expected that one of the EFPIA beneficiaries/large industrial beneficiaries may elect to become the coordinator or the project leader. Therefore to facilitate the formation of the final consortium, all beneficiaries are encouraged to discuss the weighting of responsibilities and priorities therein. Until the roles are formally appointed through a Consortium Agreement the proposed project leader shall facilitate an efficient negotiation of project content and required agreements.

The below architecture for the full proposal is a suggestion; different innovative project designs are welcome, if properly justified.

The work plan should enable the construction of a precompetitive research platform focussed on furthering our scientific understanding on how synaptopathies can elicit or contribute to brain disorders. It should focus on the development and validation of both existing and innovative translational tools and platforms to facilitate drug-discovery-targeted synaptic health. If successful, the plan should deliver knowledge and validated technologies to facilitate the delivery of promising pharmaceutical therapies for the treatment of CNS disorders linked to deficits in synapse function. All deliverables should be achieved using scientifically robust experimental studies, agreed upon with the consortium partners, and conducted across multiple sites employing both existing and novel experimental models of synapse dysfunction together with deep clinical phenotyping of defined patient populations.

The work plan must reflect the pooling of resources and expertise from academia, SMEs and industry in a fully integrated public-private partnership that will ultimately deliver a heightened understanding of the contribution of synapse dysfunction to CNS disorders together with a battery of robust, validated, decision-making preclinical and clinical platforms to facilitate drug development.

Applicants should suggest the most suitable project architecture to implement the below activities within two phases, an initial phase and a late phase of action.

**Initial phase (approx. 3 years)**

1. The consortium partners should undertake an early appraisal of all available disease models, preclinical and clinical platforms together with selected patient cohorts to prioritise activities and ensure the most effective delivery of the project objectives;

2. Cross-site characterisation of in-vivo synaptopathy disease models using the predefined platforms and technologies will be initiated and derived data will be used to identify those disease models and platforms most optimal for drug discovery efforts. Potential disease models may include but should not be limited to transgenic mouse strains expressing risk genes for psychiatric, neurodegenerative and developmental disorders;
3. Clinical protocols will be composed and regulatory/ethical approvals gained to permit initiation of recruitment for the synaptopathy phenotyping of defined patient cohorts. Recruitment will be initiated and an ad-interim analysis conducted for signal detection and power analysis determination. These assessments can include but may not be limited to:

- Demonstration of the grade of usefulness of existing PET ligands (made available from the industry consortium, namely SV2A and AMPA TARP), and 18F-FDG as markers of synapse integrity/function;
- Development and assessment of synaptic PET tracers, for example novel ligands targeting post synaptic density protein 95 (PSD-95), vesicular glutamate transporters (VGLUT1/2), excitatory amino acid transporter-2 (EAAT2) or others to be determined;
- Assessment of clinical imaging modalities such as functional magnetic resonance imaging, magnetoencephalography and diffusion tensor imaging for relevance and suitability to detect synaptic alterations in patient cohorts and correlation of these readouts with above synaptic function PET tracers;
- Assessments of clinical EEG measurements employing a battery of paradigms that induce cognitive or other functional event-related brain potentials or coherence;
- Develop behavioural and synaptic imaging marker phenotyping for selected CNS disorder animal models to be able to establish translation between synaptic marker and behavioural endpoint.

4. Interactions with patient groups and regulatory bodies will be initiated to discuss appropriate development paths forward for novel therapies targeting synaptopathies.

**Late phase (approx. 2 years)**

Based on successful implementation of the above activities, the remaining two years will focus on:

1. Cross-site profiling of existing and novel therapies believed to address positively synaptopathy in the defined in-vivo disease models using the battery of preclinical platforms;

2. Clinical evaluation of novel PET ligands targeting pre- and post-synaptic proteins, for example synaptic density protein 95 (PSD-95), vesicular glutamate transporters (VGLUT1/2), excitatory amino acid transporter-2 (EAAT2) or others to be determined;

3. Determination of pharmacological sensitivity of defined clinical assessment battery using existing chemical entities thought to modulate synaptic function, for example ketamine or others to be determined.

**Work package 1 – Clinical work streams**

The goals of this work package will be as follows:

- Characterisation of existing PET ligands, namely SV2A and AMPA TARP and 18F-FDG, which may prove to be useful markers of synapse integrity/function. This work package could also be extended to the development of novel PET ligands targeting PSD-95, VGLUT1/2, EAAT2 for example. Other imaging modalities such as functional magnetic resonance imaging, magnetoencephalography and diffusion tensor imaging should also be considered based on their relevance and suitability to detect synaptic alterations;

- Development of novel behavioural assessments (cognitive, motor and psychosis/mood related) and EEG measurements, that better reflect synaptic function and alterations as defined by translatable synaptic PET markers.

**Industry consortium contribution:**

- Clinical and disease area experts with access to patient cohorts;
- Clinic ready PET ligands (Synaptic Vesicle Glycoprotein 2A (SV2A) and α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid Transmembrane Regulatory Proteins (AMPA TARP)) in addition to PET chemistry support for novel ligand development;
- Clinical expertise in trial design, implementation and regulatory support;
Access to preclinical synaptopathy models (AD, PD, MDD, Schizophrenia, Autism).

Expected applicant consortium contribution:
- Clinical and disease area experts with access to patient cohorts;
- PET ligand development expertise;
- Rodent PET capabilities;
- Imaging experts;
- Clinical trial operation and execution;
- Expertise in clinical data management and clinical statistics;
- Expertise in patient advocacy and engagement, privacy and ethical considerations;
- Regulatory expertise and experience in development and qualification of novel end-points;
- Data management.

Work package 2 – Preclinical work streams

The goals of this work package will be as follows:
- Cross-site characterisation of in-vivo synaptopathy disease models using the predefined platforms and technologies. Potential disease models may include, but should not be limited to, transgenic mouse strains expressing risk genes for psychiatric, neurodegenerative and developmental disorders;
- Technologies and platforms development and assessment: these may include but should not be limited to electrophysiological, imaging, synaptic fluid biomarkers measurements, neurotransmitter sensors, optogenetics and behavioural platforms (cognitive, motor and psychosis/mood related).

Industry consortium contribution:
- Disease models known to demonstrate synaptic dysfunction linked to neurodevelopmental, psychiatric and neurodegenerative disorders including but not exclusive to transgenic mouse strains expressing risk genes for neurodevelopmental, psychiatric and neurodegenerative disorders;
- Access to technologies, know-how and protocols including but not limited to:
  - Electrophysiological (MEA, LTP/LTD, EEG, ERP’s);
  - Imaging (calcium, high content, immunohistochemical, autoradiography, 2-DG);
  - Synaptic fluid biomarkers measurements (SNAP-25, GAP-43, LAMP-2 for example);
  - Microdialysis, neurotransmitter sensors and optogenetics;
  - Behavioural platforms (cognitive, motor and psychosis/mood related).
- Commercially available and development tool compounds.

Expected applicant consortium contribution:
- Academics and SMEs with disease understanding of neurodevelopmental, psychiatric and neurodegenerative disorders;
- Expertise in disease model generations and characterisation;
- Preclinical imaging, electrophysiology and fluid biomarker experts;
Work package 3 – Management, dissemination, stakeholder interaction, data & knowledge management and sustainability

The goals of this work package will be as follows:

- Management & coordination
- Communication and dissemination
- Interaction with stakeholders including regulators

Please see the Call conditions for further details. In particular, applicants are reminded that full proposals must contain a draft plan for the exploitation and dissemination of the results.

- Data and knowledge management

To ensure adherence to the legislation concerning protection of personal data, controlled access digital repositories and data governance need to be considered. Proposals should use well-established data format and content standards for data collection and data management in order to ensure interoperability to quality standards and optimal use of IMI resources. Preferably existing standards should be adopted. Should no such standards exist, consideration should be given to adapt existing standards in collaboration with a data standards organisation (e.g. CDISC). Only if no existing useable standards exist in any format should consideration be given to developing new standards in collaboration with relevant bodies to ensure that any new standards are planned to become the de facto standard for any relevant future projects.

In addition, technical solutions (tools, data repositories etc) for data storage, management, analysis or visualisation should always re-use existing solutions where possible in preference to the development of new resources. For instance, many scientific data needs are now well served by well-established open source or commercial solutions, which should be identified in the application, with appropriate budget projections. This could include such areas as (but not limited to): Electronic lab notebooks, biological assay data analysis tools, ‘omics data storage and analysis etc.

The applicants should provide in their short proposals a brief description of the data and knowledge management plan that will be further detailed in the data management plan in the full proposal. They should also ensure resource and budgetary planning for data management and include a deliverable for an initial Data Management Plan (DMP) at month 6 at the latest into their proposal (see guidelines of FAIR data management in H2020).

- Sustainability

A plan for aspects related to sustainability, facilitating continuation beyond the duration of the project should also be proposed.

Industry consortium contribution:
Support to communication/dissemination and project management activities.

Expected applicant consortium contribution:
Applicants are expected to contribute to the implementation of all of the above activities.
References


