A LOSS OF TIME?

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14.10.21 Juf

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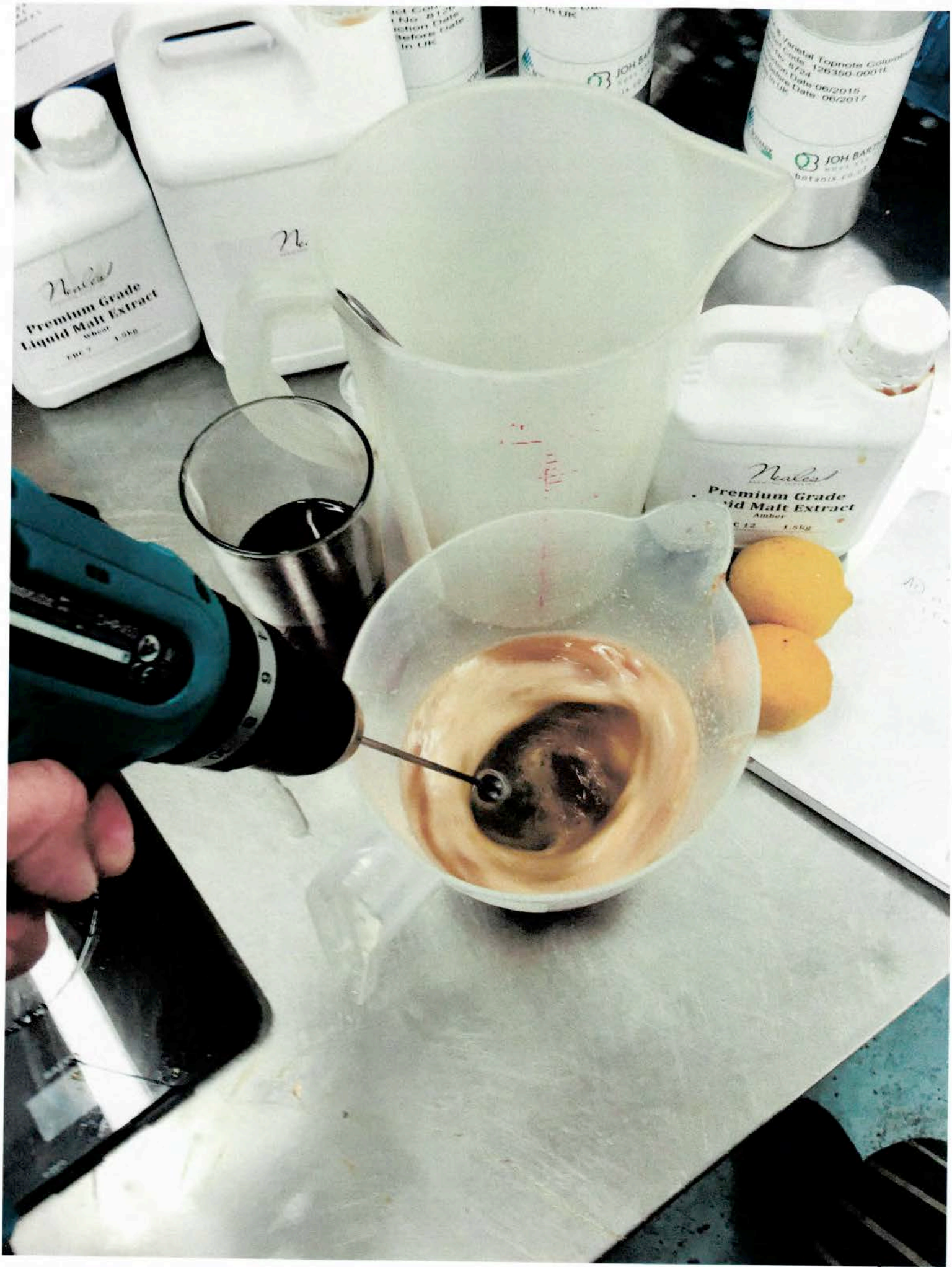
Rhizome Analysis : Tumeric 2016

Rhizome Analysis: Galangal

2016

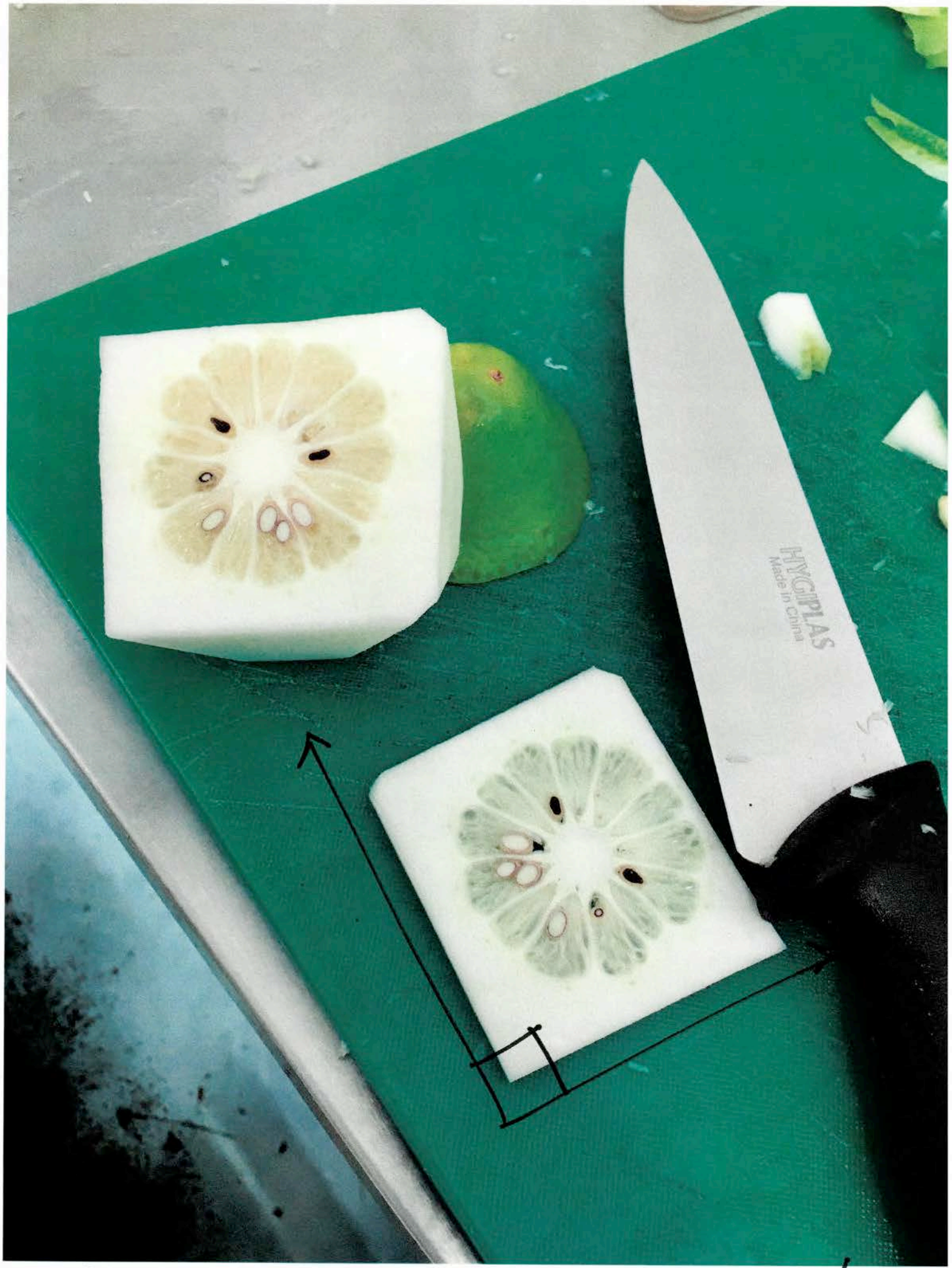
Rhizome Analysis: Ginger 2016

Centrifugal Pump Assemblage 2016

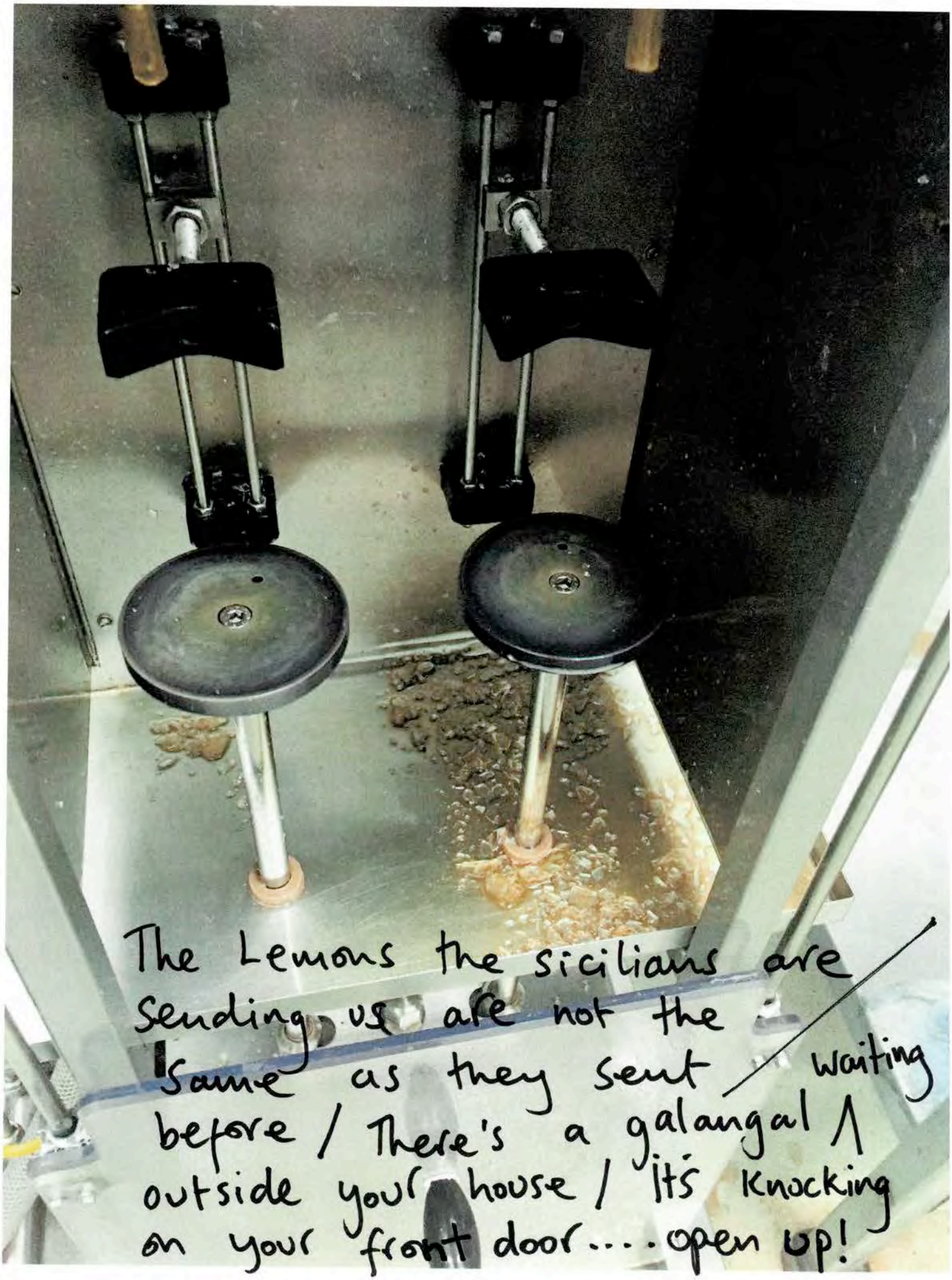


Malt Mixture Methodology

2016



Plato Leman 2016


The Lemons the sicilians are
sending us are not the
same as they sent waiting
before / There's a galangal /
outside your house / It's knocking
on your front door.... open up!

Malady , Malaise , Maladaptation

Abbas Zahedi

C3 Neuropharmacology

Time to get 'Movin'

Introduction:

Parkinson's disease (PD) is a debilitating progressive neurodegenerative disorder which affects at least 1% of the global population. Furthermore it has been documented that every general practitioner in the United Kingdom treats multiple patients with the disease, and there is yet to exist a general consensus on the most its most effective therapy[1]. Symptoms of PD are typified by the slowness or loss of movement (akinesia), muscle stiffness, rigidity, and resting tremors affecting the extremeties. The underlying cause of the disease is loss of neural connectivity between the substantia nigra (SN) and caudate-striatum, two brain regions which are indispensable in motor function. These neurones contain dopamine (DA), and it is the resultant decrease of DA due to neural death which leads to the onset of disease[2]. Establishing a cause for PD is yet to be done, thus the main focus of clinicians treating patients with PD is symptomatic drug therapy. Most treatments revolve around the neurobiology of DA, with levodopa (L-DOPA) being the most common. L-DOPA is a precursor for intracellular synthesis of DA, by the enzyme dopa-decarboxylase. Because this enzyme only exists in dopaminergic neurones, as the progressive nature of the disease persists there will ultimately be no site of action remaining for L-DOPA. This has serious implications in the use of this drug for treating patients with PD. The fluctuations which occur between the positive and negative effects of L-DOPA mark the beginning of the end for the usefulness of this drug. This results in a process clinically reffered to as the 'wearing-off' period, and after this period choice of therapy is very limited indeed[3]. Thus it is obvious why there is pertinent need for new treatments with higher efficacy and much more prolonged effect. Recently a newly discovered drug termed 'Movin' caused a stir in the research community, due to its positive effects against parkinsonian symptoms, when tested in an animal model of the disease[8]. This was the MPTP-treated primate model, which is known to be the most accurate reproduction of the human disease in animals. This model involves immunoreactive degeneration of neurones relevent to PD in the brain and has contributed significantly to developments in the fight against PD[4]. The data shows that a modest dose of the drug (when compared to current drugs) is of use in reducing PD related symptoms such as akinesia and rigidity, though the mechanisms involved in doing so remain to be elucidated. Therefore I wish in this proposal to study the effects of Movin as a potential treatment for PD and determine a putative understanding of its mechanism for action. I will achieve this by using rodent models of the disease (presented below); which like the MPTP-primate model, have proven to be of great value, and were amongst the first animal models to accurately recreate parkinsonian symptoms.

Preliminary Results: THE LOSS OF MOVEMENT = A LOSS OF TIME ?

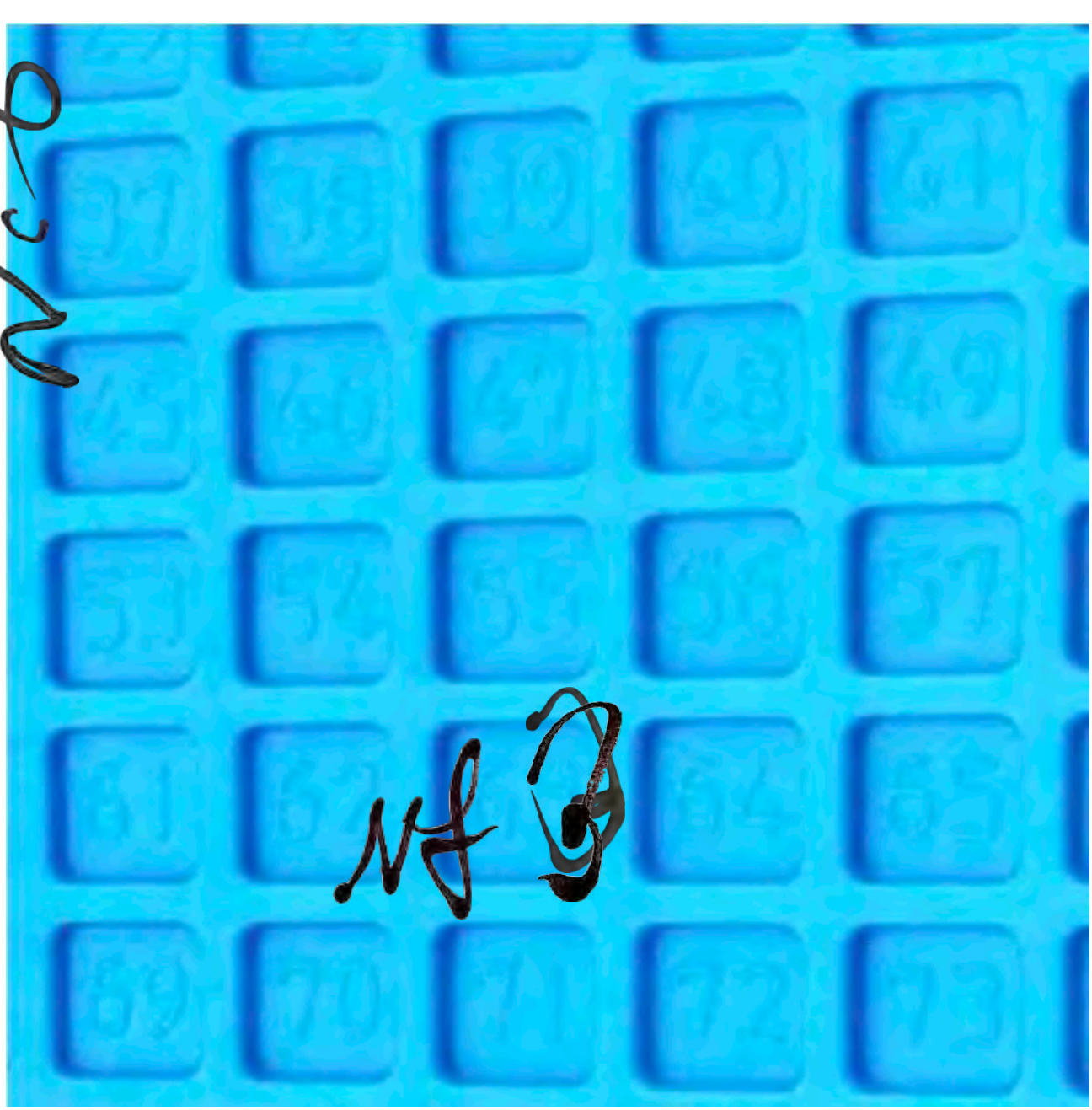
Experiment 1:

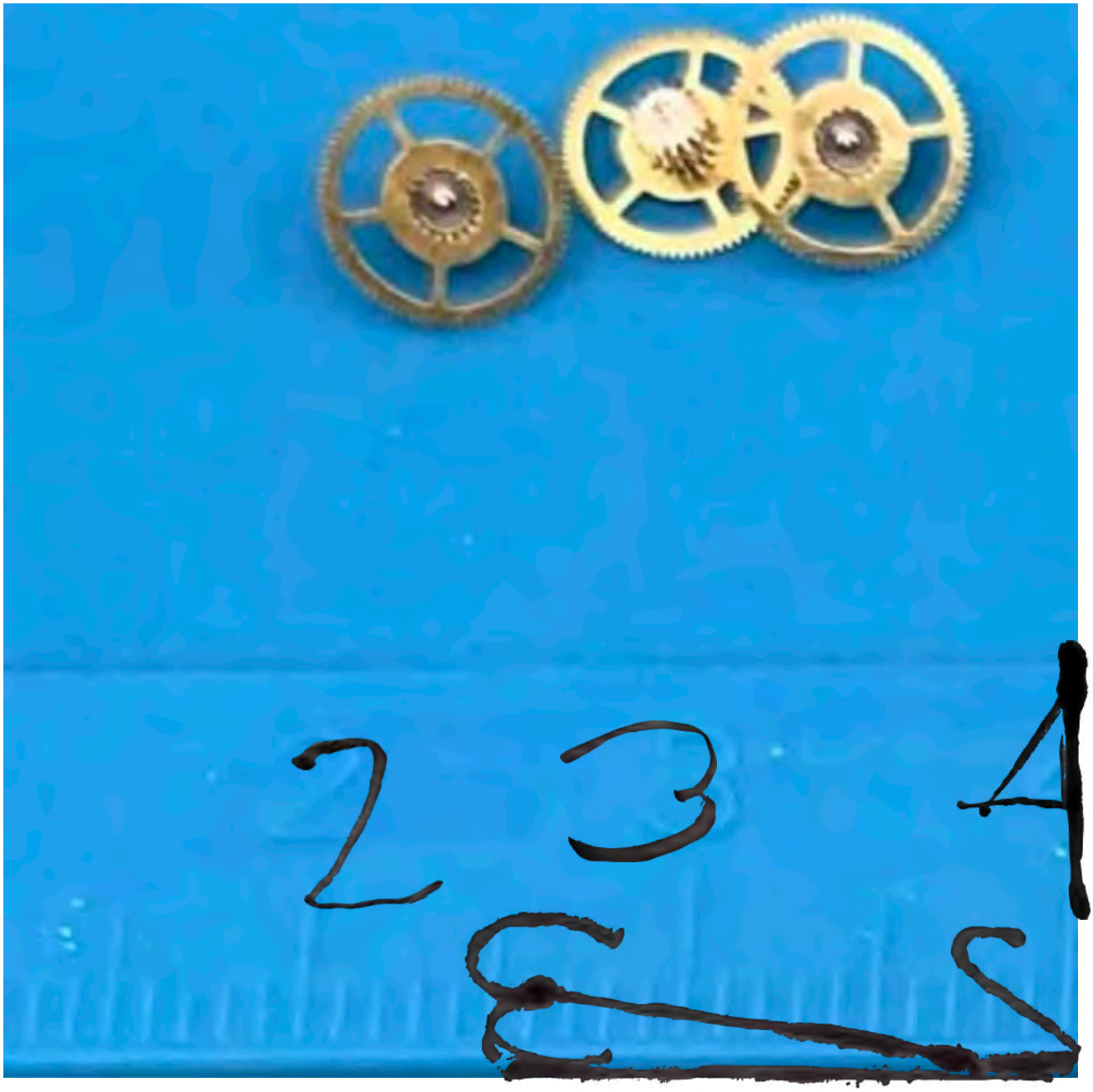
The reserpine rodent model of Parkinson's disease is a well established test for the effect of drugs against PD. Reserpine binds to vesicular monoamine transporter 2 (VMAT-2) and prevents storage of DA in vesicles, this causes DA to build up in the cytosol of the neurone. Independent of exocytosis DA is released into the synaptic cleft and causes a brief augmentation of dopaminergic function, after which the depleted neurones no longer partake in DA transmission. The effects of reserpine closely mimic the decline in motility associated with PD. Addition of L-DOPA reverses the effects of reserpine as is shown in the following experiment:

The activity of mice running on a rotating treadmill was recorded after administration of the aforementioned drugs. Mice were tested against a negative control mouse which did not receive any drug. The activity of each mouse was determined by the number of rotations resulted from its walking or running in the treadmill. We quantified these rotations by use of magnets which passed each other upon every full rotation, and total number of rotations was saved by a computer linked to the apparatus. The drug dependent models we used in this study are listed below:

Malady , Malaise , Maladaptation

Alberto Vallejo

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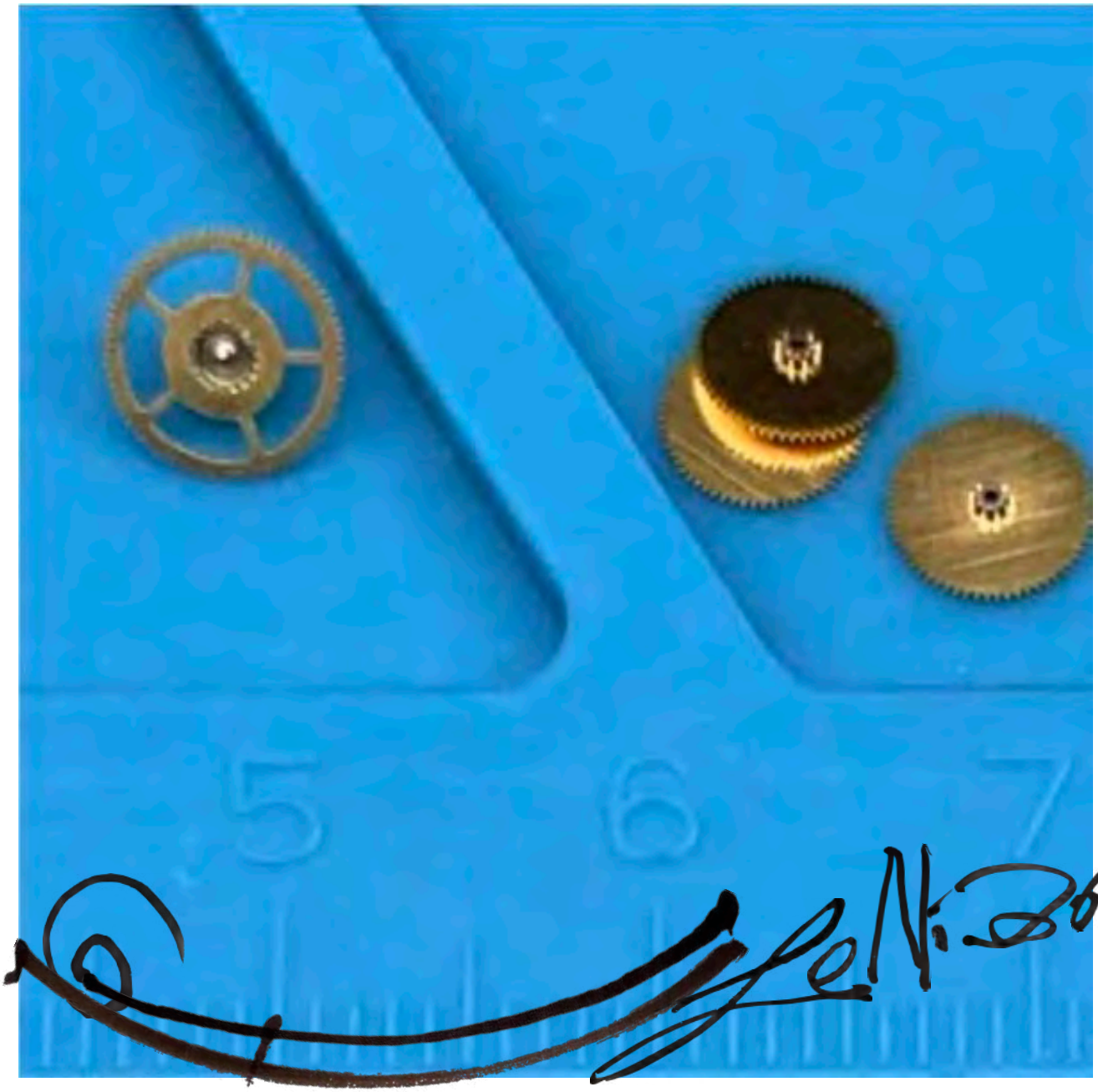

Abbas Zahedi's interdisciplinary practice blends contemporary philosophy, poetics, and social dynamics with sound, sculpture, and other performative media. With an emphasis on how personal and collective histories interweave, Zahedi makes connections whenever possible with those around, in proximity to, or involved with the particular situations upon which he focuses.

Alberto Vallejo is an artist and curator based in Madrid where he co-curates the independent art space Yaby since 2017.

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