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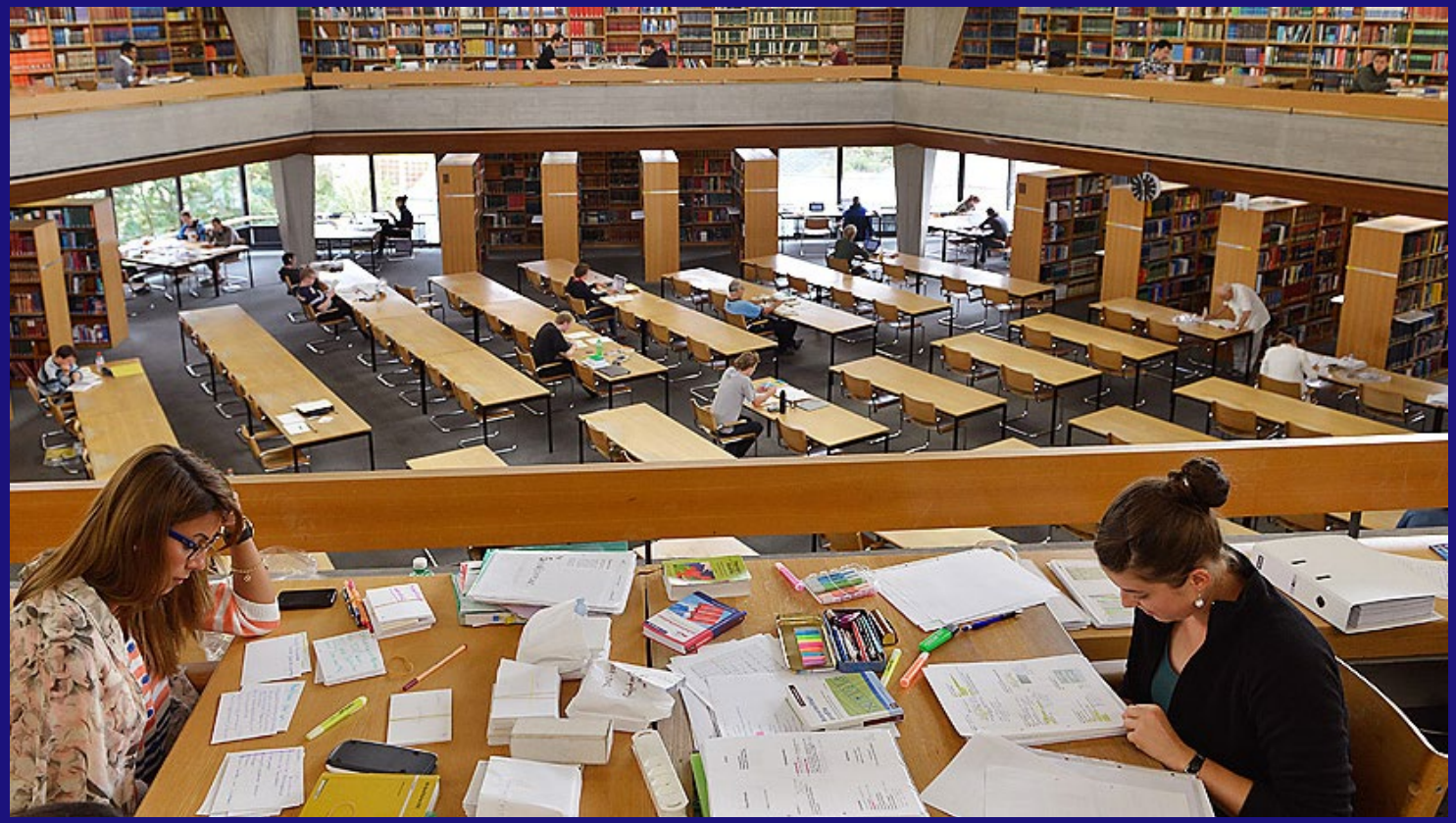

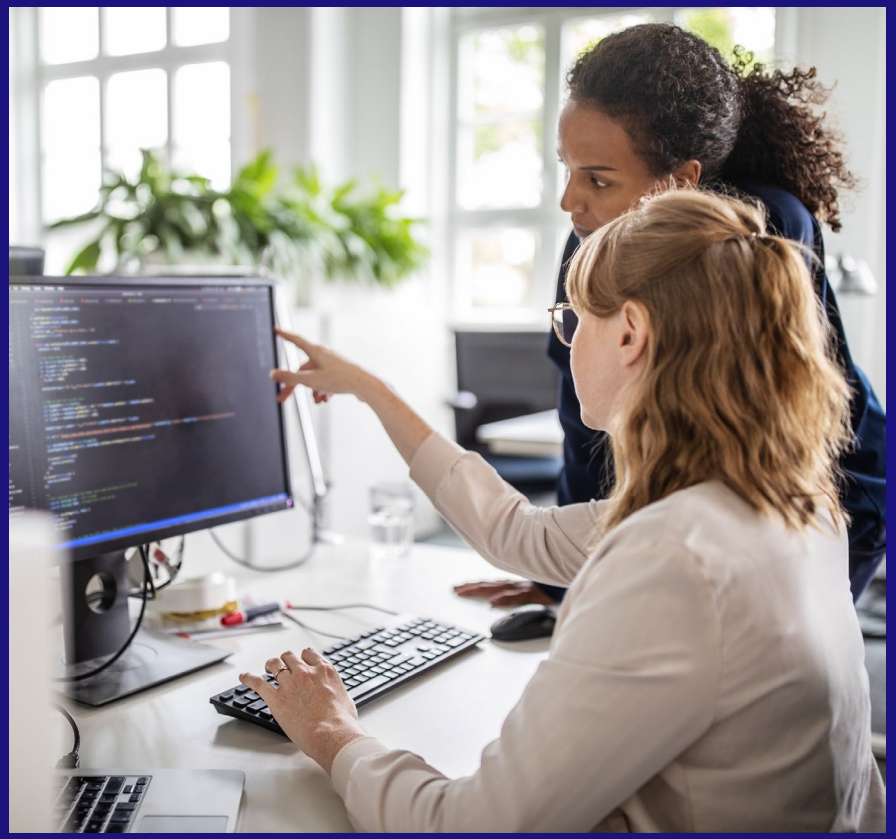

Presentation title

V. *Method of preparing a cheap Subſtitute for Oil Paint, as durable as that prepared with Oil, and free from any bad Smell. By M.* LUDICKE. *From the* Bibliotheque Phyſico-économique, 1792.

IT often happens that people do not chooſe, or cannot employ oil-painting in the country, either becauſe it does not dry ſoon enough and has an inſupportable ſmell, or becauſe it is too dear. M. Ludicke employed, with the greateſt ſucceſs, the following method for painting ceilings, gates, doors, and even furniture.

The Proceſs.

Take freſh curds, and bruiſe the lumps on a grinding-ſtone or in an earthen pan or mortar with a ſpatula. After this operation, put them in a pot with an equal quantity of lime well quenched and become thick enough to be kneaded; ſtir this mixture well, without adding water, and you will ſoon obtain a white-coloured fluid, which may be applied with as much facility as varniſh, and which dries very ſpeedily. But it muſt be employed the ſame day, as it will become too thick the day following.

Ochre, Armenian bole, and all colours which hold with lime, may be mixed with it, according to the colour which you wiſh to give to the wood; but care muſt be taken that the addition of colour made to the firſt mixture of curds and lime may contain very little water, elſe the painting will be

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RESEARCH PAPER

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Starvation after infection restricts enterovirus D68 replication

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ABSTRACT

Enterovirus D68 (EV-D68) is a respiratory pathogen associated with acute flaccid myelitis, a childhood paralysis disease. No approved vaccine or antiviral treatment exists against EV-D68. Infection with this virus induces the formation of autophagosomes to enhance its replication but blocks the downstream autophagosome- lysosome fusion steps. Here, we examined the impact of autophagy induction through starvation, either before (starvation before infection, SBI) or after (starvation after infection, SAI) EV-D68 infection. We showed that SAI, but not SBI, attenuated EV-D68 replication in multiple cell lines and abrogated the viral-mediated cleavage of host autophagic flux-related proteins. Furthermore, SAI induced autophagic flux during EV-D68 replication and prevented production of virus-induced membranes, which are required for picornavirus replication. Pharmacological inhibition of autophagic flux during SAI did not rescue EV-D68 titers. SAI had the same effect in multiple cell types, and restricted the replication of several medically relevant picornaviruses. Our results highlight the significance of autophagosomes for picornavirus replication and identify SAI as an attractive broad-spectrum anti-picornavirus strategy.

Abbreviations: BAF: bafilomycin A_1; CCCP: carbonyl cyanide m-chlorophenylhydrazone; CQ: chloroquine; CVB3: coxsackievirus B3; EV-D68: enterovirus D68; hpi: hour post-infection; MAP1LC3/LC3: microtubule associated protein 1 light chain 3; MOI: multiplicity of infection; NSP2B: nonstructural protein 2B; PV: poliovirus; RES: resveratrol; RV14: rhinovirus 14; SAI: starvation after infection; SBI: starvation before infection; SNAP29: synaptosome associated protein 29; SQSTM1/p62: sequestosome 1; TFEB: transcription factor EB.

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Autophagic flux; EV-D68; picornavirus; SAI; SBI

Introduction

First discovered in 1962, the incidence of enterovirus D68 (EV-D68) infection has been on the rise over the past decade. Since its discovery, EV-D68 has caused clusters of acute respiratory illness in multiple countries, and recent shreds of evidence implicate its infection with a rare polio-like neurological disease, acute flaccid myelitis, in children making EV-D68 a respiratory pathogen of public health importance [1–3]. Currently, there are no effective antivirals or prophylactic vaccines against EV-D68 infection. Therefore, understanding how EV-D68 interacts with the machinery of its host cells could open avenues for therapeutic interventions against the enterovirus.

EV-D68 belongs to the *Piconarviridae* family of viruses, which includes important human and animal viruses such as poliovirus, coxsackievirus B3, enterovirus A71, hepatitis A virus, and Seneca Valley virus, among others [4,5]. The viral genome is 7500 nucleotides long and encodes a single polyprotein that is processed by viral proteases to four structural proteins (VP1, VP2, VP3, and VP4), which enwrap the autophagosomes [6]. Indeed, multiple lines of evidence have shown that picornaviruses utilize autophagosomes for RNA replication [7–9].

Macroautophagy (hereafter autophagy) is a conserved catabolic process that targets long-lived proteins and damaged cytoplasmic organelles to the lysosomes for degradation. The cellular process can also target microorganisms, including bacteria and viruses, for degradation through xenophagy, forming an essential part of the immune response [10]. Autophagy can be induced by several stress stimuli, including amino acid starvation. Autophagy begins with the formation of the phagophore, which expands and elongates through two ubiquitin-like conjugation systems: the conjugation of ATG5, ATG12, and ATG16L1; and the addition of phosphatidylethanolamine (PE) to MAP1LC3B/LC3B (microtubule associated protein 1 light chain 3 beta) to form the autophagosome, which then fuses with the lysosomes to degrade the cargo [11]. The lipidation of LC3B (conversion of soluble LC3B-I to the membrane-bound LC3B-II) and the degradation of the receptor protein, SQSTM1, is widely used to monitor autophagy [12].

If I have seen further it is by standing on the shoulders of giants.

Isaac Newton

The value chain


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What is the likely shape of the library of the Future?
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Thank you for listening

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