Study	Link	Species	General microscopy method	Visualization method specifics	PMI range	Longest PMI reported	Sample size	Brain region	Disease studied, if any recorded	Structural Feature	Outcome	Relevant text	Any significant/substantial correlation of a feature with PMI reported?
Albrechtsen 1977a	https://pubmed.n	Human	Light microscopy	Morphological staining with H&E; histochemical staining for naphthylamidase	9 to 271 hours	271 hours	70	Cerebellum	Multiple conditions	Histochemistry for naphthylamidase	No reported association between PMI and histochemical appearance of naphthylamidase (LNAse). Low pH is associated with necrosis of the granule layer.	"The distribution of LNAse activity was the same in all normal cerebella (49 cases)" ... "Regardless of the cause of death LNAse activity was found in all cases throughout the granular layer without any demonstrable variation in the loss of activity in the central and peripheral parts. No cases were found without enzyme activity." ... "[T]he LNAse activity was preserved with unchanged activity even many hours after death. Lysosomal enzymes similarly share a n unusual resistance to autolysis"	No
Albrechtsen 1977b	https://pubmed.n	Human	Light microscopy	Morphological staining, for example with H&E	6 to 83 or more hours (83 hours is the highest PMI listed, but the full range not reported)	83 hours	1000	Cerebellum	Not recorded	Presence and severity of necrosis of granule cell layer of the cerebellum	Seems to be a tendency for an increase in degree of granule cell necrosis with increasing PMI, but it is not statistically significant. Note that necrosis is mostly graded based on properties of the cell nuclei.	"Table 4 shows the incidence of necrosis according to severity, related to the interval between death and autopsy. There would seem to be a tendency for increased number of necroses with ioncreasing interval, but the difference is not statistically significant."	Yes
Ansari 1976a	https://pubmed.n	Human	Electron microscopy	Staining with osmium tetroxide, uranyl acetate, and lead citrate	8 to 48 hours, compared to biopsy tissue	48 hours	7	Frontal lobe white matter	Not recorded	Myelin	Increase in myelin lamellae splitting during the PMI with no break in continuity.	"On electron microscopic examination, unfrozen white matter obtained at autopsy (8 h post-mortem) showed lamellar separation similar to that described in cerebral edema (14). This observation of post-mortem lamellar separation secondary to edema is consistent with the reported increase in water content of the brain (approximately 10%) that occurs during the time interval between death and autopsy (3). This unfrozen autopsy material, however, showed no break in the continuity of the separated myelin lamellae."	Yes
Arnold 1995	https://pubmed.n	Human	Light microscopy	Morphological staining with cresyl violet	4 to 30 hours	30 hours	24	Multiple brain regions	Schizophrenia	General neuronal morphology	No significant correlation between PMI and neuron size in multiple brain regions.		No
Beach 1988	https://pubmed.n	Human	Light microscopy	Horseradish peroxidase filling	3 to 11 hours	11 hours	3	White matter underlying the cerebral cortex	Clinical diagnosis of dementia	Neuronal morphology	Worse labeling with horseradish peroxidase at PMIs of longer than 8 hours. However, no loss of structural integrity under light microscopy at the longer PMIs.	"In both cortical and brainstem preparations, postmortem intervals longer than 8 h resulted in less extensive labelling." ... "This may be due to loss of structural integrity within the tissue, although this was not evident at the light microscopic level. In fact, tissue morphology was very good throughout these blocks."	Yes
Bédard 2004	https://pubmed.n	Human	Light microscopy	Confocal microscopy, immunostained for markers or proliferation and immature neuronal state	4 to 24 hours	24 hours	10	Olfactory bulb	No "clinical or pathological signs of neurological or psychiatric disorders"	General neuronal immunostaining	No major difference found resulting from the PMI range.	"[D]espite significant inter-individuals variations with regard to age, postmortem delay and cause of death, the overall immunostaining pattern of the olfactory bulb was found to be strikingly similar in the 10 human subjects used in the present study."	No
Benes 1991	https://pubmed.n	Human	Light microscopy	Nissl staining	Mean 13.9 ± 2.8 (control group); 32.8 ± 19.5 (schizophrenia and no mood disturbance group); 34.5 ± 14.8 (schizophrenia and mood disturbance group)	Not recorded	23	Hippocampus	Schizophrenia	Neuronal morphology	No significant correlation between PMI and neuron size.	Table 6	No
Benes 1992	https://pubmed.n	Human	Light microscopy	Glutamate immunoreactivity of vertical fibers	Mean 19.1 ± 32.9 hours (control group); 19.6 ± 20.1 hours (schizophrenia group)	Not recorded	32	Anterior cingulate cortex	Schizophrenia	Neuronal processes	No significant correlation between PMI and fiber density.	"The postmortem interval was similar for the control and schizophrenic groups, as were the fixation interval and the hypoxia index. The age of the schizophrenic subjects (48.5 ± 23.0 years), however, was lower than that for the control cases. Simple linear regression analyses indicated that there was not a significant relationship between any of the confounding variables and either small- or large-caliber glutamate-immunoreactive fibers (Table 2), suggesting that these factors, particularly age, do not account for the differences in fiber density observed in the schizophrenic group"	No
Benes 2001a	https://pubmed.n	Human	Light microscopy	GluR5,6,7 immunostaining	4.5 to 74.6 hours	74.6 hours	39	Hippocampus	Schizophrenia	Dendrite morphology	No significant correlation between PMI and pyramidal cell dendritic density.	"The potential confounding effects of age and PMI do not appear to account for the differences in IR dendrites noted in the schizophrenic group, because all three groups were matched with respect to both of these variables. Moreover, there were no significant correlations between these two variables and the density of IR dendrites in each of these sectors."	No
Benes 2001b	https://pubmed.n	Human	Light microscopy	Nissl staining	1.9 to 66.5 hours	66.5 hours	33	Anterior cingulate cortex	Schizophrenia and bipolar disorder	Cellular morphology	No correlation between PMI and morphologically-identified cell density across groups. Subgroup correlations were found (PMI is positively associated with non-pyramidal neuron density in the bipolar group and negatively associated with glial density in layer II in the control group); however, there was no adjustment for multiple comparisons.	"When the three groups were combined, correlation analyses revealed that there were no significant relationships of age or PMI with the density of PNs, NPs, or glial cells in any of the layers. Similarly, when the data were broken down according to diagnostic groups, there were also no significant relationships between these two potential confounds and the density of PNs and NPs in the schizophrenic subjects. For the PMI, the density of glial cells in the control subjects showed a significant negative correlation in layer II (r = −.57; p = .05). For the bipolar group, however, there was a significant positive correlation (r = .80; p = .007) between the density of NPs and PMI."	Yes
Blair 2014	https://pubmed.n	Human	Light microscopy	Immunostaining for amyloid beta and morphologically staining with H&E	3 to 40 hours (mean PMIs ranging from 11-16 hours in different age groups)	40 hours	92	Hippocampus	Multiple conditions	General neuronal morphology	No correlation of PMI with intraneuronal amyloid beta staining.	"This prompted further examination of the cases to determine whether iAbeta presence was correlated with postmortem interval (PMI), gender, or presence of AD. Variable iAbeta immunoreactivity was noted in both very short and very long PMI subsets (Fig. 3). Two biopsy samples were strongly positive for iAbeta immunoreactivity yet some cases with a PMI of 24 h also had strong iAbeta immunoreactivity. Analysis of PMI for all cases examined showed no correlation between PMI and iAbeta immunoreactivity intensity (p>0.1). Gender was also found to not be a factor in i iAbeta immunoreactivity (p>0.1). Evaluation of hematoxylin and eosin stained sections, revealed that all samples were well preserved with ideal cellular morphology and showed no evidence of tissue disruption or infection." Also Figure 3.	No
Booze 1993	https://pubmed.n	Human	Light microscopy	Immunostaining for tyrosine hydrolase	1 to 6.5 hours	6.5 hours	10	Multiple regions	Multiple conditions	Tyrosine hydrolase immunoreactive axons	Staining for fine varicose axons was lost with increasing PMI.	"A pronounced and significant decrease in axon type 2 occurred as a function of increasing postmortem interval." ... "Type 2 axons were characterized as being very fine and highly varicose".	Yes
Boros 2017	https://pubmed.n	Human	Light microscopy	Golgi-Cox and 3d reconstruction	3 to 78 hours	78 hours	41	Dorsolateral prefrontal cortex	Alzheimer's disease	Dendrite morphology	Dendritic spine density is not associated with PMI.	"Linear regression analysis indicated that spine density was independent of sex or postmortem interval and that spine density changes within disease states were not associated with age"	No
Brück 1996	https://pubmed.n	Human	Light microscopy	Morphological staining and in situ tailing protocol	4 hours to 3 days	72 hours	13	Multiple brain regions	Pontosubicular neuron necrosis	Presence of apoptotic markers	No PMI effect on evidence of cellular apoptosis.	"The number of cells labelled in the IST reaction did not depend on the interval between death and autopsy nor on the duration of formalin fixation (Table 2). Even with brains fixed for 30 or 47 days (cases 8 and 6), positive results were obtained in the IST reaction which corresponded to the number of apoptotic cells evaluated morphologically"	No
Buell 1982	https://pubmed.n	Human	Light microscopy	Morphological staining with Golgi-Cox and rapid Golgi techniques	6 to 28 hours	28 hours	10	Multiple brain regions	Multiple conditions	General neuronal morphology and dendritic morphology	No variation of neuronal morphology, including dendritic morphology, with PMI when using Golgi-Cox staining.	"Almost invariably, the Golgi-Cox method impregnated a large number of neurons which were fairly evenly distributed. Dendritic extent varied greatly within each section, from cells with rich, apparently normal dendritic trees to cells with clearly atrophic trees. Examples of each type, as well as of cells which appeared intermediate between the two extremes, were numerous. Tissues prepared by the rapid Golgi method gave a very different picture. Impregnation was often patchy with large areas devoid of impregnated cells. The cells which did impregnate were predominantly those with grossly atrophic trees or aberrant morphology. Our findings do not vary according to age, mental status, or postmortem time of sampling."	No
Chan-Palay 1986	https://pubmed.n	Human	Light microscopy, electron microscopy	Immunostaining for neuropeptide Y	Comparison of surgical biopsy cases (n = 19) to autopsy cases (n = 12) with a PMI of 8.5 to 36 hours	36 hours	31	Cerebral cortex	Multiple conditions	Neuropeptide Y immunostaining	Worse preservation in the autopsy tissue than the surgical biopsy tissue. Axons can usually be traced, but there are more varicosities they are more irregular in size and shape. Dendrites are shorter, thicker, and more contorted. Neuronal cell bodies are reported to be "generally clearly immunoreactive". Despite having this worse reported preservation, they also report that "NPY-immunoreactivity is well preserved in neural structures for considerable periods after death".	"In the surgical biopsy specimens immunostained with anti-NPY serum, the neurons of the cerebral cortex are displayed in their entirety (Fig. 1–d). The perikarya are completely filled with reaction product, the dendrites are long and slender, and thin varicose axons can be traced from their initial segments for long distances, sometimes even up to 3 mm. The axonal plexuses in the neighboring neuropil have numerous fine varicose axons and the whole forms a network of intricate delicacy." ... "Even the best material obtained from postmortem con- trol brains successfully tested with NPY pales by compari- son with the surgical specimens. Although optimal post- mortem material can be good, and neuronal cell bodies are generally clearly immunoreactive, the dendrites appear somewhat foreshortened and thicker and unusually tor- tuous. The most pronounced difference is in the axonal plexuses. Axons from neurons can usually be followed. The plexuses in the neuropil are also abundant but not as deli- cate in appearance. Perhaps the most distinct difference is that the varicosities or boutons on axonal twigs are larger - 2-4 times the size of those in surgical specimens, are more irregular in size and shape, and often have intervari- cose segments that are difficult to distinguish. Basically, axons are thicker, there are shorter single segments, vari- cosities are thicker, the intervaricose segments are shorter and clusters of axons are coarser. The dendrites are shorter, thicker and more contorted."	Yes
Chandana 2009	https://pubmed.n	Human	Light microscopy	Morphological stain and immunohistochemical stains for GFAP and neurofilament	4 to 18 hours	18 hours	9	Multiple brain regions	No history of neurologic disorder	General cellular morphology	No association of PMI with changes in cell morphology or cytoarchitecture, including with immunostaining for GFAP or neurofilament.	"No major abnormalities in cell morphology or tissue integrity were noted. Immunohistochemistry with GFAP and NF did not show any significant increase in signal in FC at high PMI." ... "Histological examination of the sections from four anatomical areas at different times of post-mortem delay (9 samples) revealed essentially similar features of cytoarchitecture and density of myelin. The immunolabelling character for GFAP and NF (Fig. 8) was essentially similar at 4 and 18 h PMI"	No
Craven 2005a	https://pubmed.n	Human	Light microscopy	Nissl staining, tyrosine hydroxylase immunostaining	Average of 44.4 ± 28.7 h in controls, 57.2 ± 38.2 h in donors with a diagnosis of schizophrenia	Not recorded	33	Locus coeruleus	Schizophrenia	General neuronal immunostaining	No correlation between PMI and cell size or cell number.	"Indeed, neither time in formalin nor PMI had a detectable effect on our measures of cell size or cell number."	No
Craven 2005b	https://pubmed.n	Human	Light microscopy	Nissl staining, tryptophan-hydroxylase immunohistochemistry	Average of 46.0 ± 27.7 h in controls, 57.2 ± 38.2 h in donors with a diagnosis of schizophrenia	Not recorded	35	Dorsal raphe nucleus	Schizophrenia	General neuronal immunostaining	No correlation between PMI and cell size or 5-hydroxytryptamine immunoreactive cell profiles.	"PMI and time in formalin had no detectable effects on our measures of cell size. But whereas PMI had no effect on the number of immunoreactive profiles counted, formalin-fixation time correlated negatively and significantly with profile number. ... In the case of anti-tyrosine-hydroxylase staining, there was no suggestion of a relationship between PMI or time in formalin and optical density"	No

Study	Link	Species	General microscopy method	Visualization method specifics	PMI range	Longest PMI reported	Sample size	Brain region	Disease studied, if any recorded	Structural Feature	Outcome	Relevant text	Any significant/substantial correlation of a feature with PMI reported?
Das 2019	https://pubmed.n	Human	Light microscopy	DiI staining	10 to 28 hours	28 hours	13	Hippocampus	Sudden death	Neuronal morphology	No correlation between PMI and degradation of neuronal morphology, such as dendritic spines and axon boutons.	"Figure 2 shows a representative set of neurone and dendritic segments from several brains. Note that despite being from tissue fixed 11–18 hours postmortem, we do not observe blebbing, neurite fragmentation, or other obvious hallmarks of cell death, hypoxia, or ischemia (Figure 2). Though we cannot exclude that any postmortem interval leads to changes in neuron morphology, the neuron morphology in our samples appears well preserved and the secondary dendrites are a consistent caliber with even DiI labeling and diffusion." ... "In contrast, our DiI-based methodological pipeline generates high quality dendritic spine labeling in human tissues with a longer postmortem delay, thus far we tested up to 28 hours postmortem. When labeled, the neurons do not show obvious morphological signs of hypoxia, ischemia, or damage cause neuron such as blebbing, fragmentation, or poor diffusion of DiI (Fig 1e), suggesting good membrane integrity and relatively healthy tissue. Here, we report a mean CA1 apical spine density of 2.21 spines/µm of dendrite with a range from 1.36 to 3.04 spines/µm in adult human brain. These spine densities in neurons labeled from tissue fixed 10–28 hours postmortem is strikingly consistent with previously reported spine densities of control human CA1 neurons in tissue fixed 2–3 hours postmortem (Merino-Serrais et al., 2013) and to the average CA1 spine density found in non-human primates optimally preserved by perfusion."	No
Del Bigio 2000	https://pubmed.n	Human	Light microscopy	Morphological staining with H&E, without or without LFB	0.5 hours to 80 hours, including 3 surgical cases (0.5 to 2 h delay to fixation)	80 hours	33	Multiple regions	Multiple conditions	Glia morphology	Increased cell swelling as the PMI increases.	"On the basis of our observations and other published data, we conclude that human glia, both astrocytes and oligodendrocytes, rapidly take up plasma proteins from the extracellular space of the injured brain" ... "[T]he pronounced swelling with eosinophilia is exaggerated by postmortem delay of fixation. Whether this represents an active process that continues in a devitalized environment, or a passive response by cells with abnormally high soluble protein content in the cytoplasm is unknown."	Yes
Garey 1998	https://pubmed.n	Human	Light microscopy	Rapid Golgi staining	4 to 120 hours	120 hours	24	Layer III of temporal and frontal neocortex	Schizophrenia	Dendrite morphology	No correlation between PMI and dendritic spine density of pyramidal neurons.	"There was a significant inverse correlation (p<0.01) between age and spine density in the non-schizophrenic controls but the correlation was not significant for postmortem interval and spine density, whether this was calculated by parametric (Pearson) or non-parametric (Spearman) tests (table 3 and fig 3)."	No
Geiger 2006	https://pubmed.n	Human	Light microscopy	TUNEL staining	Up to 48 hours	48 hours	24	Multiple brain regions	HIV encephalopathy	Presence of apoptotic morphology	No correlation between PMI and the detection of apoptotic cell morphology.		No
Gibson 1979	https://pubmed.n	Human	Electron microscopy	Morphological stain, embedded in Araldite	0 to 69 hours	69 hours	45	Frontal pole or middle temporal gyrus	Dying in hospital from causes unlikely to lead to neurological complications	Synapse	Vacuolization increased up to approximately 33 hours, then stays steady or decreases. Causes significant compression of tissue and artifactual distortion, but no obvious structural degeneration of membranes.		No
Gittins 2004	https://pubmed.n	Human	Light microscopy	Cresyl violet or NeuN staining	8 to 75 hours	75 hours	16	Anterior cingulate cortex	Not recorded	Neuronal morphology	No correlation between PMI and neuronal morphology parameters.	"Neuronal density, size and shape did not correlate, in either Nissl or NeuN stained material, with post-mortem interval (−0.39<R<0.27; all P>0.13) or age (−0.25<R<0.31; all P>0.19)." ... "Post-mortem interval, even over an extended range, showed no correlations with neuronal density, size or shape, suggesting that this factor is relatively unimportant."	No
Glausier 2019	https://pubmed.n	Human	Electron microscopy	Osmium and uranyl acetate staining	0 to 24 hours	24 hours	30	Dorsolateral prefrontal cortex	Psychiatric disorder	Synapse	Significant negative correlation with the number of PSD identified and PMI, but no significant correlation with PSD length and PMI; no significant correlation with total neuronal profiles identified and PMI.	"PMI was significantly negatively correlated with number of PSD."	Yes
Gos 2014	https://pubmed.n	Human	Light microscopy	Immunostaining for quinolinic acid	10 to 72 hours	72 hours	25	Hippocampus	Schizophrenia	Microglia density	Decreased number of quinolinic acid-immunoreactive microglial cells in the right CA1 subregion of the hippocampus with increased PMI.	"Analysis of the potential confounding factors on the test results revealed no significant influence of age, duration of disease or psychotropic medication. Autolysis time was negatively correlated with the numerical density of QUIN-immunoreactive microglia in the right CA1 (controls: rPearson = 0.592, p = 0.043, p = 0.016; schizophrenia: rPearson = 0.661, p = 0.014). However, the above described diagnostic group effect for CA1 was confirmed by ANCOVA with the covariate "autolysis time" (schizophrenia versus controls; left p = 0.024, right p = 0.008)."	Yes
Huh 2001	https://pubmed.n	Human	Light microscopy	all-bdp1 immunostaining	4 to 22.8 hours	22.8 hours	6	Multiple	Free of recognized neurological disease	General immunostaining	No correlation between PMI and immunostaining distribution for all-bdp1.	"The overall level of immunopositivity or all-bdp1 in the adult human brain was sparse, although focally positive cells were identified in every region. There was no correlation of the frequency of all-bdp1 positive cells with the postmortem interval or with the rare cells noted above that appeared to display perimortem anoxic/ischemic change. A semi-quantitative analysis of neuronal immunopositivity for all-bdp was conducted by visual survey, with score 1 representing immunopositive neurons up to 25% of the neuronal population and score 4 representing immunopositive neurons of more than 75% of the neuronal population. The highest score was noted in the cerebellum (mean score, 2.33; Table 1). There was no correlation between calpain-cleaved spectrin and the postmortem interval (P % 0.6041, r % 20.232, by Spearman rank correlation). ... In the cerebellum, moderate to strong and occasionally focal immunoreactivity in Purkinje cells was consistently found (Fig. 2A). As with the pyramidal neurons, only the soma and apical dendrites of cerebellar Purkinje cells were immunopositive (Fig. 2B)"	No
Hunziker 1977	https://pubmed.n	Human	Light microscopy	Morphological staining via histochemistry for alkaline phosphatase	6.5 to 8.5 hours and 30 hours	30 hours	2	Multiple brain regions	Not recorded	Capillary morphometry	Capillary network is reported to be relatively resistant to postmortem changes, with no change in capillary diameter. However, there is a change in volume fraction, length, surface-to-volume ratio, and number of capillaries per test area in the longer PMI case.	"In contrast to the experiment the human investigation revealed no in- fluence on the above-mentioned stereological parameters at two postmor- tem times. Although volume fraction, length, surface-to-volume ratio and number per test area of the parietal, temporal and occipital lobe (Tab. 3) yield tendencies similar to those recorded in the experiment, the capillary network of the human cerebral cortex appears to be relatively resistent to postmortem changes. The fact that capillary diameter remains unchanged postmortem provides encouragement to continue further stereological in- vestigations of aging human brain" ... "As in the animal experiment, the capillary diameter of the human cortex is not affected by different postmortem times. The individual diameter values of the parietal lobe (postcentral gyrus) do not differ much from each other (Fig. 3). Except for the frontal region, the stereological behaviour of the human parameters is similar to the significant differences between the measurements intravitam and 22 hours postmortem in the animal experiment (Tab. 2): volume fraction, length per unit cortex volume and number of capillary fragments per measuring field tend to decrease, whereas the surface-to- volume ratio is moderately increased (Tab. 3). The minimal capillary distances vary only weakly in dependence on the two different postmortem times."	Yes
Huttenlocher 1979	https://pubmed.n	Human	Electron microscopy	Morphological staining with phosphotungic acid	0 to 35 hours	35 hours	21	Middle frontal gyrus	No known neurologic disease or severe prolonged hypoxia prior to death	Synapse	Synapse counts were stable for 35 h PMI. Compared to perfusion fixed experimental tissue, synaptic structures are less clearly demarcated, but this does not appear to be a correlated with PMI in their sample. (Reported difference from expected results basen on perfusion fixation versus immersion fixation, but not based on the PMI within the sample.)	"Samples from 21 brains, at ages ranging from newborn to 90 years, were studied. Synaptic profiles were clearly evident in postmortem cerebral cortical tissue, although their definition was often somewhat less sharp than in perfusion-fixed experimental tissue (Fig. 1A). The presynaptic projections and postsynaptic bands appeared somewhat more diffuse and less sharply demarcated, and the intracleft lines were not always demonstrable. However, these features did not seriously interfere with the ability to enumerate synaptic profiles. Synapse counts appeared to be stable for 35 h postmortem (Fig. 2), no relationship being demonstrable between calculated synaptic densities and the length of time that elapsed before the tissue was fixed in glutaraldehyde"	No
Ikemoto 2003	https://pubmed.n	Human	Light microscopy	Immunohistochemistry for aromatic l-amino acid decarboxylase (AADC)	2 to 30 hours	30 hours	18	Midbrain, striatum	Schizophrenia	Aromatic l-amino acid decarboxylase positive neurons	Decreased staining of aromatic l-amino acid decarboxylase (AADC), including decreased number of AADC neurons, with longer PMI.	"Some brains in which the PMI was more than 15 h showed weak AADC stainability. The number of AADC-positive neurons in the striatum per section had a tendency to decrease in the case with longer PMI"	Yes
Ikuta 1963	https://pubmed.n	Human	Light microscopy	Morphological staining, for example with H&E	0 to 3 to more than 18 hours	18 hours	162	Cerebellum	Not recorded	Presence of necrosis of granule cell layer of the cerebellum	Increased incidence of necrosis of the granule cell layer with increasing PMI.	"There is a steady increase in the incidence of the lesion as [the PMI] increases."	Yes
Jacobs 1993	https://pubmed.n	Human	Light microscopy	Silver staining	5.5 to 32 hours" (cases longer than 24 hours included if they were in good condition histologically)	32 hours	20	Superior temporal gyrus	"Neurologically normal"	Dendrite morphology	Dendritic spine architecture is well preserved in brains with longer autolysis times. Note that there is a quality selection bias in this study.	"[A]nalysis revealed no relationship between autolysis time and TDL (r[4001 = 0.03; NS). Removing the effect of autolysis on the age-TDL correlation with a partial correlation coefficient (see below) reconfirmed that autolytic effects on TDL were minimal. The tissue examined did not exhibit the autolytic changes (e.g., irregular varicose enlargements, constriction of dendrites, incomplete impregnation, and loss of spines) described by Williams et al. ('87). Photomicrographs of Golgi preparations from three subjects (F40, M56.2, F79) with differing autolysis times (29, 10.5, and 8 hours, respectively) are presented in Figures 2 and 3" ... "Tissue was excluded if the autolysis time was unknown, or if there were any signs of cerebral atrophy, cerebral edema, chronic illness with central nervous system involvement, and so on."	No
Kay 2013	https://pubmed.n	Human	Electron microscopy	Staining with osmium tetroxide and uranyl acetate	Up to 100 hours	100 hours	Not recorded	Multiple brain regions	Multiple conditions	General cellular morphology	Reported effective electron microscopy studies with tissue up to 100 hours PMI, as long as the brain is primarily in cold storage with a temperature of 4-6°C. It seems they are suggesting that there is a PMI effect if the brain tissue is not cold storage, but this is not explicit.	"We have managed to conduct very effective EM studies on tissues retrieved from donors with long post mortem intervals, up to 100 hours. In our experience a key element in tissue preservation for ultrastructure analysis is post mortem cold storage of the cadaver, with cold storage in a mortuary of around 4–6°C significantly reducing structural degradation."	Unclear

Study	Link	Species	General microscopy method	Visualization method specifics	PMI range	Longest PMI reported	Sample size	Brain region	Disease studied, if any recorded	Structural Feature	Outcome	Relevant text	Any significant/substantial correlation of a feature with PMI reported?
Kitamura 2005	https://pubmed.n	Human	Light microscopy	MAP2 immunostaining	11 h to 3 days (all less than 1 day in normothermic group)	72 hours	20	Hippocampus	Hypothermia. No "neurodegenerative disease, intracranial hemorrhage or brain injuries that would induce MAP2 disruption before their lethal event"	General neuronal immunostaining	PMI-associated decline in immunostaining of MAP2 in CA1-subiculum, with less rapid decline in immunostaining in CA2-4 and dentate gyrus, and less rapid/remarkable PMI-associated decline in immunostaining in brain donors who had hypothermia prior to death.	"With lengthening postmortem intervals, loss of MAP2 immunoreactivity progressed in most of the CA1-subiculum neurons" ... "In the brains of a PMI of 11–16 h (cases H1–3), MAP2 immunoreactivity was preserved in the dendrites of a large number of the CA1-subiculum neurons (Figs. 1a and 2a), although cytoplasmic accumulation of MAP2 was more evident in the area adjacent to the stratum oriens than in other areas (Fig. 2a). Intensity and localization of MAP2 staining had altered less in brains with a PMI of 1.5 d (cases H4–5) (Fig. 2b) than in the normothermic group (Fig. 2c and d). In brains with a PMI of 2–3 d (cases H6–8), the CA1 neurons showed decreases in the number of immunopositive dendrites and intense cytoplasmic immunoreactivity, although the changes were less remarkable." ... "On the other hand, we observed that postmortem disruption of MAP2 progressed remarkably in the CA1-subiculum region of normothermic brains with increasing PMI, although there were less postmortem changes in MAP2 immunostaining in the CA2–4 regions and the dentate gyrus" ... "[H]ypothermia attenuated the progress of postmortem alterations in MAP2 immunoreactivity in the CA1-subiculum region where MAP2 disruption was more remarkable than other regions"	Yes
Kolomeets 2005	https://pubmed.n	Human	Electron microscopy	Staining with osmium tetroxide, uranyl acetate, and lead citrate	3 to 9 hours	9 hours	19	Hippocampus	Schizophrenia	Dendrite morphology	No significant correlation between dendritic spine morphology and PMI.	"Multiple regression analysis did not reveal any significant interactions among the age or PMI and thenumber of spine heads, and postsynaptic spineheads, and Vv of spines per MFT."	No
Kolomeets 2007	https://pubmed.n	Human	Electron microscopy	Staining with osmium tetroxide, uranyl acetate, and lead citrate	Mean 5.96 ± 1.1 (control group), 6.6 ± 1.9 (schizophrenia group)	Not recorded	18	Hippocampus	Schizophrenia	Synapse density	No correlation of PMI with the number of synapse contacts detected on mossy fiber terminals.	"Our research design did not indicate that the observed reduction of Nv synapses formed by MFTs was due to age or PMI. No significant relation between the parameters measured and age or PMI were found by correlation analysis"	No
Krause 2016	https://pubmed.n	Human	Electron microscopy	Morphological staining with osmium tetroxide, uranyl acetate, and phosphotungic acid	18 to 50 hours	50 hours	Not recorded	Anterior cingulate cortex	Not recorded	General cellular morphology	Myelin sheaths have network-like splittings. Other structures such as Von Economo Neurons, microtubules, neurofilaments, synapses, and vesicles were reported to be well-preserved. No reported worsening of ultrastructure with PMIs greater than 24 hours.	"In human pathology, the post-mortem interval is a relevant factor for tissue preservation. It is known that degenerative alterations in the cytoplasm of glial cells and within the axoplasm can occur within 24 h at 4 °C, and especially mitochondria are known to be vulnerable to degeneration. The same degenerative effects already appear after 6 h at a storage temperature of 25 °C (Hukkanen and Röyttä, 1987). In our study, the tissue had a mean post-mortem interval of 31.89 ± 9.649 h and showed the typical ultra-structural alterations of network-like splitting of the inner part of the myelin sheath (Fig. 3A), as described by Hukkanen and Röyttä (1987). However, other structures such as microtubules, neurofilaments, synapses, vesicles and mitochondria were well preserved (Fig. 3B–D). In particular, entire neurons such as VENs, which are currently of most interest in investigations of psychiatric disorders, could be depicted clearly in a collage of merged micrographs (Fig. 3E)." ... "Summarising, in ultra-structural studies on human post-mortem tissue it is more important to use tissue with similar post-mortem intervals than similar storage times. Furthermore, it is possible to undertake ultra-structural investigations on specimens with high post-mortem intervals above 24 h."	Yes
Kreczmansk 2007	https://pubmed.n	Human	Light microscopy	Morphological stain, Nissl stain (gallocyanin)	6 to 76 hours	76 hours	26	Multiple brain regions	Schizophrenia	Neuronal density	No assocation of PMI with neuronal density in any brain region, but there was a significant assocation with total putamen volume and total neuron neuron in the putamen with PMI.	"The post-mortem interval had a significant effect on the volume of the putamen [F(1) ¼ 9.553, P ¼ 0.004] and the total neuron number in this brain region [F(1) ¼ 6.899; P ¼ 0.012] (see Fig. S1 in the Supplementary online material)." Also Table 3.	Yes
Kreczmanski 2005	https://pubmed.n	Human	Light microscopy	Immunostaining for collagen IV, which is present in the basal membranes of microvessels	6 to 88 hours (they note that the 88 hour brain was a case who died of suicide and was stored at 4°C)	88 hours	26	Multiple brain regions	Schizophrenia	Blood vessels	No significant effect of PMI on capillary length density.	"[P]ostmortem interval, and fixation time had no significant effect on any of the investigated parameters."	No
Lesnikova 2018	https://pubmed.n	Human	Light microscopy	Morphological stain with H&E, immunohistochemical stains with S100 and vimentin	1 to 14 or more days	336 hours	40	Not recorded	Not recorded	General cellular morphology	Increase in cell nucleus markers of autolysis in increasing PMI, with loss of cells being the most extreme grade. Decreased immunostaining properties of vimentin and S100 as the PMI increased past 3 days.	"Brain tissue had a mean decomposition score of 1.11/median of 1.10 in group A, 1.53 /1.35 in group B, 2.72/1.40 in group C, and 3.77/5.00 in group D". Also Figure 4.	Yes
Lindenberg 1956	https://pubmed.n	Human	Light microscopy	Morphological stain with cresyl violet or thionine	8 to 46 hours	46 hours	45	Multiple brain regions	No known internal disease	General neuronal morphology	In the cases with sudden death, vacuolization, homogenization, or ischemic changes were present, associated with the postmortem interval. In cases preceded by an agonal hypoxia of 1-7 hours, the morphology was much better maintained, without vacuolization, shrinkage, or swelling, despite the degree of PMI.		Yes
Liu 1950	https://pubmed.n	Guinea pig	Light microscopy	Morphological stain with thionine and a technique to visualize myelin sheaths	1 to 13 hours (immersion fixation group)	13 hours	16	Multiple brain regions	Malnutrition	General cell morphology	Lighter myelin staining, some swollen neurons, other shrunken neurons, vacuolization, perineuronal and perivascular retraction spaces, compared to perfusion fixation immediately at the time of death.	"The 16 animals that died from inanition before they could be killed by the perfusion-fixation technic, and whose brains were fixed by immersion in solution of formaldehyde U.S.P. (1:4), showed neuronal changes not unlike many of those that have been described by previous investigators. Sections of the brain prepared by the method for myelin sheaths stained lighter than similar sections from the animals that had been killed by perfusion and lighter than sections from the control animals. Similarly, the sections stained by the technic for Nissl substance appeared lighter. Microscopic examination demonstrated marked generalized changes in all parts of the brain. Some nerve cells were considerably shrunken; others were swollen. The Nissl body patterns were clouded, and in some instances there appeared to be almost complete chromatolysis. Many nerve cells were vacuolated. The perineuronal and perivascular spaces were much larger than those in sections from the control animals. Even in the specimens that were fixed by immersion within one hour after death, such changes were widespread. The similarity between this picture and that obtained in human brain material removed at autopsy and fixed by immersion in solution of formaldehyde U.S.P. was striking."	Yes
Lopes 2008	https://pubmed.n	Human	Light microscopy	Immunostaining for ferritin	3 to 20 hours	20 hours	9	Temporal and frontal cortices	Not recorded	Microglial morphology	No correlation of the morphology of microglia with PMI, although there was an increase in nonspecific background staining after 8 hours PMI.	"At a PMI of 3 h, ferritin immunoreactive microglial cells exhibited a mostly branched morphology (Fig. 6A), although distal processes were largely devoid of fine ramifications. Similar morphological characteristics were observed at longer PMIs (Figs. 6B–D), and most ferritin-positive microglia in these tissues displayed deramified and beaded processes as described earlier (see Figs. 2 and 3). Instances of deramified microglial cells remained constant from shortest to longest PMI. However, nonspecific background staining was found to increase after a PMI of 8 h."	Yes
Lucassen 1997	https://pubmed.n	Human	Light microscopy	ISEL labeling	1 to 20 hours	20 hours	65	Multiple brain regions	Alzheimer's disease	General cell morphology	No major apoptotic cellular morphology resulting from the PMI.	"No apoptotic morphology, such as nuclear condensation, membrane blebbing, or apoptotic bodies, was observed in any of the brain areas studied."	No
Maloku 2010	https://pubmed.n	Human	Light microscopy	Morphological stain, Nissl stain (cresyl violet)	20 ± 6.1 h for nonpsychiatric subject group, 22 ± 5.0 h for schizophrenia group, 20 ± 9 h for bipolar disorder group	Not recorded	54	Cerebellum	Schizophrenia, bipolar disorder, controls	Neuronal density	No correlation of PMI with Purkinje cell density.	"Our mixed models on Purkinje neuron linear density failed to find that background variables had an effect on Purkinje neuron linear density, except for the difference in diagnosis. This included age [F1,421 = 1.51, nonsignificant (NS) when treated as continuous variable; F2,420 = 1.61, NS when treated as categoric variable], gender (F1, 421 = 2.61, NS), PMI (F1,421 = 2.75, NS), hemisphere (F1,421 = 1.56, NS), brain pH (F1, 421 = 3.32, NS), suicide (F1,421 = 0.31, NS), age of onset of illness (F1,214 = 0.24, NS), other cause of death (F4,410 = 2.09, NS), or type of medication (F3,188 = 1.01, NS)."	No
Mori 1991	https://pubmed.n	Human	Light microscopy	IgG and laminin immunostaining	5 to 40 hours	40 hours	28	Frontal or temporal lobes	Alzheimer's disease	Blood vessels	Increase in leakage of immunoglobulin out of blood vessels in the PMI.	"The extent and severity of leakage in both the aged and Alzheimer brains tended to increase with the age of the patient and with the interval between death and autopsy."	Yes
Mori 1992	https://pubmed.n	Human	Light microscopy	Laminin immunostaining	5 to 40 hours	40 hours	26	Frontal cortex	Not recorded	Blood vessels	Increased laminin immunostaining/blood vessel visualization with increased postmortem delay.	"In untreated sections, the areas possessing immunoreactivity tended to be larger as the postmortem delay was prolonged. This was especially notable in 2 cases fixed after prolonged postmortem periods (21 to 40 h), in which moderate immunoreactivity, weaker than that of unfixed frozen sections, was observed throughout the sections. " Also Figure 5.	Yes
Müller 2021	https://pubmed.n	Human	Light microscopy	Morphologically stained with combined cell and fiber staining according to Nissl (cresyl violet) and Heidenhain–Woelcke	4 to 164 hours	164 hours	24	Medial habenula	Heroin use disorder	Neuronal density	No correlation of PMI with neuronal density.	"[N]euronal cell numbers were reduced in the MHB of heroin-addicted subjects (395,966 ± 184,178 vs. 644,149 ± 131,140; p < 0.001). These findings were not significantly confounded by age and duration of autolysis." ... "Neurons of heroin addicts were of normal appearance. The cytomorphological analysis by an experienced neuropathologist (CM) revealed no alterations of Nissl bodies or other cytostructural changes in neurons of heroin addicts." ... "Accordingly, no significant influence of age (MHB: F(1,21) = 0.015, p = 0.902; LHB: F(1,21) = 0.420, p = 0.524) and duration of autolysis (MHB: F(1,21) = 0.212, p = 0.650; LHB: F(1,21) = 0.213, p = 0.649) was observed on relative habenular volume differences. Furthermore, these factors showed no significant interaction with diagnosis-related differences of MHB neuronal cell numbers (age: F(1,21) = 0.312, p = 0.582; autolysis: F(1,21) = 0.108, p = 0.746)."	No
Nogami 1999	https://pubmed.n	Human	Light microscopy	Hsp70 immunoreactivity	5 to 56 hours	56 hours	34	Medulla oblongata	Not recorded	General neuronal immunostaining	No significant correlation in proportion of neuronal cytoplasmic staining for Hsp70 with the PMI.	"Hsp70 immunoreactivity was present in the cytoplasm of some neurons of the XII, X, Cun, and Oli (Fig. 1), while no neuronal staining was observed in the negative control with the non-specific rabbit immunoglobulins. There were statistically significantly fewer neurons with positive cytoplasmic hsp70 immunoreactivity in the Oli than in the XII, X, or Cun (p<0.05 for XII, p<0.01 for X or Cun, Kruskal-Wallice test and Dunn's test, n=34, Fig. 2). There was no statistically significant correlation between the AMI or PMI, and the percentage of positive cytoplasmic hsp70 immunoreactivity in any of the nuclei studied (Pearson's test, n=33 for AMI, 30 for PMI)."	No

Study	Link	Species	General microscopy method	Visualization method specifics	PMI range	Longest PMI reported	Sample size	Brain region	Disease studied, if any recorded	Structural Feature	Outcome	Relevant text	Any significant/substantial correlation of a feature with PMI reported?
Nogami 2000	https://pubmed.n	Human	Light microscopy	Cathepsin D immunostaining	5 to 151 hours	151 hours	45	Hippocampus	Not recorded	General cytoplasmic immunostaining	No correlation between PMI and immunostaining distribution for Cathepsin D.	"CA1 neurons showed strong diffuse cathepsin D immunoreactivity in the cytoplasm (Figure 1A) in all but one case." ... "In one case of chest injury in a traffic accident, CA1 neurons had low cathepsin D immunoreactivity, in which the neurons morphologically showed an ischemic change of cytoplasmic shrinkage (Figure 1B and C). The percentages of positive, cathepsin D immunoreactive cells are shown in Table 1. Virtually all CA1 neurons were positively stained in the majority of cases, in which the percentage of positive neurons was calculated as 100%. No statistically significant correlation was observed between cathepsin D immunoreactivity and the antemortem interval between injury and death (AMI), or the postmortem interval between death and autopsy (PMI) (Pearson's test)." ". Hill et al. (1997) have shown that global ischemia by two-vessel occlusion in rats resulted in an alteration in the distribution of cathepsin B in hippocampal CA1 neurons from a lysosomal pattern to a more intense label redistributed into the cytoplasmic region. Since some postmortem intervals were inevitable in our study, our autopsied specimens showed intense distribution of cathepsin D in the whole cytoplasm" Figure 1 and 2. "Although lysosomal enzymes including cathepsin D are activated in postmortem necrosis (Lindenberg 1982), there was no clear change in the distribution of cathepsin D immunoreactivity, such as extracellular leakage, or loss of cytoplasmic immunoreactivity. Our results show that cathepsin D immunoreactivity in CA1 neurons is not a useful tool to evaluate neuronal degeneration or postmortem changes."	No
Ohm 1994	https://pubmed.n	Human	Light microscopy	Intracellular filling with Mini-Ruby (MR) dye	7 to 50 hours	50 hours	35	Not recorded, although hippocampus and entorhinal cortex are among the regions studied	Multiple conditions	Neuronal morphology	No correlation with PMI in intracellular morphology dye filling. "The systematic examination of autopsy conditions using M R indicated that neither age (P > 0.1; Kolmogo- roff-Smirnoff's ~ ~ = 0.4263) nor postmortem delay (P > 0.1; Kolmogoroff-Smirnoff's 8 = 0.1864) of individuals which gave good fillings differed significantly from those with bad fillings."	"The systematic examination of autopsy conditions using [Mini Ruby] indicated that neither age (P > 0.1; Kolmogoroff-Smirnoff's ~ ~ = 0.4263) nor postmortem delay (P > 0.1; Kolmogoroff-Smirnoff's = 0.1864) of individuals which gave good fillings differed significantly from those with bad fillings."	No
Panayotacopoulou 2002	https://pubmed.n	Human	Light microscopy	Tyrosine hydroxylase immunostaining	2 to 39 hours	39 hours	38	Hypothalamus	No neurological, psychiatric, or endocrinological disease	General neuronal immunostaining	No correlation between PMI and immuostaining distribution of tyrosine hydroxylase.	"This remarkable variability concerned the number and distribution of TH-IR perikarya as well as the intensity of the immunohistochemical reaction and appeared to be related neither to the sex or age of the subjects nor to the postmortem interval or staining procedures. The interindividual differences were evident in samples obtained from both sources after different postmortem delays and fixation or storage times in formalin using any one of the three immunohistochemical procedures on vibratome or paraffin sections."	No
Pejhan 2020	https://pubmed.n	Human	Light microscopy	Immunostaining for MeCP2 and BDNF	Surgical biopsies cases (n = 16) and autopsy brain samples, PMI range of 6 h to 5 days	120 hours	32	Multiple	Rett syndrome	General cellular immunostaining	PMI of >48 hours is associated with variable and inconsistent immunostaining for MeCP2. BDNF is more stable, with a slight decrease in immunostaining with PMI of greater than 3 days.	"To study the impact of PMD on MeCP2 staining, we performed MeCP2 immunolabelling on surgical and autopsy neocortex samples (Figure 1B–D and Fig- ure S4). MeCP2 immunoreactivity in two operative samples that were immediately fixed was similar to the operative hippocampus samples; and almost all the cells were positive (Figure 1B). Fixation delay of <48 h was associated with minor loss of MeCP2 immunoreactivity (Figure 1C). However, longer delays were accompanied by variable and inconsistent MeCP2 immunostaining (Figure 1D)." ... "Furthermore, we found that BDNF immunoreactivity was relatively stable across the range of PMD studied here. The cytoplasm of perivascular astrocytes and/or endothelial cells was labelled in immediately fixed tissue (Figure S9A–C). Fixation delay of >3 days was associated with a slight decrease in the intensity of labelling (Figure S9K–M). ... Immunostaining for BDNF in the human brain tissue array. BDNF shows higher stability with post mortem delay compared to MeCP2. A slight decrease is observed in the intensity of staining for longer intervals (fixation after 3 days). Glial cells (green arrow); endothelial cells (red arrow); Purkinje cells (purple arrow); x 400 magnification." ... "MeCP2 immunoreactivity in two operative samples that were immediately fixed was similar to the operative hippocampus samples; and almost all the cells were positive (Figure 1B). Fixation delay of <48 h was associated with minor loss of MeCP2 immunoreactivity (Figure 1C). However, longer delays were accompanied by variable and inconsistent MeCP2 immunostaining" ... "Three samples of human brain tissue array immunostained for MeCP2. Human cortex with the minimum delay to fixation (<1 h) shows complete immunoreactivity for MeCP2 (B). Delay in fixation (31 h and 5 days shown) is accompanied by variable immunostaining of all cell types"	Yes
Pennington 2008	https://pubmed.n	Human	Light microscopy	Morphologically stained with Nissl-staining (cresyl violet)	23.7 ± 9.9 h in unaffected controls, 33.7 ± 14.6 h in schizophrenia group, 32.5 ± 16.1 h in bipolar disorder group, and 27.5 ± 10.7 h in major depression group	Not recorded	60	Insular cortex	Schizophrenia, bipolar disorder, major depression	Neuronal density, neuronal size, glial density	Longer PMI correlates with increased glial density, but not neuronal density, or neuronal soma size.	"Increasing brain pH was significantly associated with decreased layer 2 (p = 0.0003) and layer 3 (p = 0004) glial volume, and increased PMI predicted increased glial cell density (p = 0.02)." ... "Specifically, neurons were distinguished by the presence of a nucleolus and some or all of the following; euchromatin, Nissl positive cytoplasm, Nissl positive dendritic processes and an ovoid or irregularly shaped nucleus. In contrast, glial cells were characterised by their lack of nucleolus, the presence of heterochromatin, a thicker nuclear membrane and a usually smaller shape and size."	Yes
Pierri 2001	https://pubmed.n	Human	Light microscopy	Nissl staining	3.3 to 24 hours	24 hours	28	Prefrontal cortex	Schizophrenia	Neuronal size	Significant change in neuronal size during the PMI.	"Exploratory regression analyses done to assess the effects of sex, age, PMI, and tissue storage time on somal volume indicated a potential effect of PMI on somal volume, which was confirmed in formal modeling."... "Third, neuronal size may change as a function of PMI as was observed in this study"	Yes
Popova 2013	https://pubmed.n	Human	Light microscopy	SLC10A4 immunostaining	24 to 244 hours	244 hours	15	Lateral geniculate body	Alzheimer's disease	General neuronal immunostaining	No correlation between PMI and immunostaining distribution for SLC10A4.	"A high number of strong SLC10A4-IR neurons were observed in the lateral geniculate body (Fig. 1g). Vesicular perinuclear labeling together with punctate and fibrillary labeling of the neuropil was observed in both magnocellular and parvocellular cell layers, whereas staining in the koniocellular cell layers was virtually absent. Noteworthy, this staining was not altered by postmortem delay (range 24–244 h) or fixation time (range 23–46 days)." ... "One, in the section of posterior hippocampus we also had the lateral geniculate body where the neurons were strongly SLC10A4-IR and the IR was not altered by postmortem delay or fixation time, issues known to influence the outcome of IHC stains'	No
Porzionato 2008	https://pubmed.n	Human	Light microscopy	Morphological stain with haematoxylin-eosin, Nissl, Klüver-Barrera, and azan-Mallory	Up to 36 hours (average 29 ± 2.1 hours)	36 hours	32	Medulla oblongata	Multiple conditions	Presence of apoptotic markers	No correlation of apoptotic markers with PMI.	"[N]o statistically significant correlation of apoptotic indices with the DAI interval in any group." ... "Moreover, tissue fixation time was quite constant for all specimens, and the apoptotic indices did not correlate with post-mortem delay. Some authors have reported that post-mortem periods of up to 48 h do not influence in situ end-labelling in rat brain (Petito & Roberts, 1995a) and that post-mortem intervals of up to 70 h do not have significant effects on the detection of apoptosis by the TUNEL method in human brain (Adle-Biassette et al. 1995; Gelbard et al. 1995; Petito & Roberts, 1995b; Vincent et al. 1999; Cosenza et al. 2004). However, we must consider that some of the apoptotic phenomena evidenced in our study are to be ascribed to post-mortem changes."	No
Raadsheer 1993	https://pubmed.n	Human	Light microscopy	Immunostaining for corticotropin-releasing hormone and vasopressin	5 to 39 hours	39 hours	13	Hypothalamus	Multiple conditions	Neuronal morphology	No effect of PMI on corticotropin-releasing hormone staining in paraventricular nucleus cell bodies or in median eminence fibers.		No
Rajkowska 2015	https://pubmed.n	Human	Light microscopy	Immunostaining for CNP	10 to 44 hours	44 hours	36	Ventral prefrontal white matter	Major depressive disorder	Oligodendrocyte density and size	No correlation of PMI with size or density of CNP-immunoreactive oligodendrocytes.		No
Rajkowska 2017	https://pubmed.n	Human	Light microscopy	Immunostaining for the serotonin transporter (SERT), using adjacent Nissl-stained sections as a guide	10 to 27 hours	27 hours	35	Orbitofrontal cortex	Major depressive disorder	Axon length	No correlation of PMI with length of serotonin-immunoreactive axons.	"For either cohort, or a combined value for both cohorts, there were no significant Pearson's correlations between the length of SERT-ir axons and postmortem interval, time in fixative, time in ethanol, or tissue pH (Table 2)."	No
Roberts 1996	https://pubmed.n	Human	Electron microscopy	Stained with osmium tetroxide and uranyl acetate	2.5 hours to greater than 7 hours	7 hours	Not recorded	Striatum	No history of central nervous system or neurological disease	Neuronal morphology	With PMI greater than 7 hours, membrane structures (pre- and post-synaptic membranes, synaptic vesicles, and myelin) were lost or distorted and dendrites; axon terminals, and dendritic spines became bloated or irregular in shape; and extracellular spaces enlarged.	"Electron microscopic observations of postmortem human brain are possible when the tissue is obtained shortly after death and immersed immediately in cold fixative containing glutaraldehyde. Several other factors other than the PMI can affect the use of postmortem tissue for electron micros- copy, such as the agonal status of the individual and ambient temperature. The ultrastructural integrity of the tissue was compromised if the PMIs were much longer than 7 hours. As the PMI increased beyond several hours, certain structures deteriorated, rendering quantitative analysis difficult, if not impossible. For example, membra- nous structures were lost or distorted, such as pre- and postsynaptic membranes, synaptic vesicles, and myelin. The postsynaptic density became unnaturally thick, mak- ing it difficult to determine whether the synapses were asymmetric or symmetric. Profiles such as axon terminals, spines, dendrites, and mitochondria became bloated andlor irregular in contour, making assessments of size inaccu- rate. The extracellular spaces enlarged in size, making measurements of synaptic density potentially inaccurate. Extracellular space was very sensitive to PMI and was expanded in some but not all areas, even in tissue with very short PMIs (<7 hours). The tissue used in this study, however, had PMIs less than 4 hours and therefore was not afflicted by these postmortem artifacts, other than an occasional expansion in extracellular space. The cases were all of a quality that permitted quantitative analysis"	Yes
Roberts 2005	https://pubmed.n	Human	Electron microscopy	Osmium and uranyl acetate staining	3 to 8 hours	8 hours	27	Striatum	Schizophrenia	Synapses	No major difference reported based on PMI; instead, adequate structural morphology was reported in the cases with "longer" PMIs. Vacuolization increased up to approximately 33 hours, then stays steady or decreases. Causes significant compression of tissue and artifactual distortion, which may explain an apparent decrease in the number of synapses visualized, but there is no obvious structural degeneration of membranes.	"The ultrastructural preservation was similar inboth groups and was suitable for synapse classifica-tion (Figs. 1–3). The electron micrographs shown arefrom cases with PMIs of 6 or 7 h to illustrate thequality of the preservation in the "longer" PMIs." ... "These micrographs illus-trate that the ultrastructural preservation is quite adequate todetermine synapse morphology even in this case that suffered a cer-tain amount of asphyxia due to the cause of death and had a"longer" PMI." ... Also Figures 1, 2, and 3	No

Study	Link	Species	General microscopy method	Visualization method specifics	PMI range	Longest PMI reported	Sample size	Brain region	Disease studied, if any recorded	Structural Feature	Outcome	Relevant text	Any significant/substantial correlation of a feature with PMI reported?
Roberts 2014	https://pubmed.n	Human	Light microscopy, electron microscopy	Immunostaining for EAAT1 AND EAAT2, with counter morphological stains	4 to 8 hours	8 hours	8	Frontal cortex	No history of central nervous system disease	General cellular morphology	No qualitative differences in EAAT protein staining patterns between 4 and 8 hours of PMI or comparing their postmortem samples to surgical biopsy samples.	"Our postmortem tissue used for electron microscopy was fixed in eight hours or under, so there should be minimal diffusion due to PMI. Also, the effect of PMI on a small mouse brain is likely to be much more dramatic than in the much larger human brain, meaning that the PMI could probably be much longer in humans without deleterious effects. Several studies using postmortem human tissue with PMIs equivalent to or greater than ours show no correlation between PMI and EAAT protein levels and/or show a similar distribution to that of the present study, suggesting that EAAT protein is not degraded in tissues with PMIs <20. (Ikematsu et al., 2001, Bauer et al., 2008, Shan et al., 2013, Shan et al., 2014). There were no qualitative differences in staining pattern between the brains used in the present study, where the PMIs ranged from four to eight hours. Finally, the ultrastructural data in human hippocampal resections (Bjørnsen et al., 2007) and cortical biopsies (Melone et al., 2011), which have no PMI, are largely in agreement with our data in postmortem tissue."	No
Rosokija 2014	https://pubmed.n	Human	Light microscopy	Morphological staining with Golgi-Cox	3 to 25 hours	25 hours	157	Cerebral cortex	Not recorded	General neuronal morphology	No effect of PMI on the quality of Golgi-Cox neuron staining.	"Over 188 consecutive frontal or hippocampal blocks from over 157 human autopsy cases with postmortem intervals varying from 3 to 25 hours were stained. There were no apparent effects of postmortem interval on the quality of impregnation."	No
Rozemuller 1988	https://pubmed.n	Human	Light microscopy	Immunostaining for antihuman C3c, Clq, IgG, prealbumin and fibrinogen	2 to 24 hours	24 hours	59	Cerebral cortex	Alzheimer's disease	Immunostaining patterns for plasma proteins	No correlation between PMI and staining patterns for plasma proteins.	"Clusters of peroxidase-positive neurons and glial cells were seen in brains both with short (3-8 hours) and long (24 hours) post-mortem delay (time between death and fixation of the brain). In cases of SDAT with the same post-mortem delay, some brains showed no staining of the brain parenchyma and others showed intensive staining of neurons and glial cells in large cortical areas. Therefore, no relation was found between post-mortem delay and positivity for plasma proteins."	No
Sarnat 2010	https://pubmed.n	Human	Light microscopy	Imunostaining for the protein synaptophysin	Up to 96 hours	96 hours	162	Multiple brain regions	Fetal and neonatal brains	Axons, Synaptophysin immunostaining	The sharp definition in axons and around neurons is lost after 24-48 hours in synpatophysin immunostained tissue. Decline of synpatophysin immunoreactivity after 48 hours. More rapid decline in choroid plexus than in grey matter.	"The sIR was preserved for more than 96 hours postmortem, even in severely autolytic brains." ... "In cases of 24 to 48 hours of postmortem autolysis before fixation of the brain, the gray matter tissue showed only a mild diffuse loss of sIR, but the sharp definition in axons and around neurons was lost. The sIR remained reliable for routine autopsy examinations up to 72 hours between death and fixation of tissue, but sIR was lost in highly macerated brains with several days of autolysis." Also Figure 6.	Yes
Scheff 1990	https://pubmed.n	Human	Electron microscopy	Staining with osmium tetroxide, uranyl acetate, and lead citrate	Up to 13 hours	13 hours	18	Cerebral cortex	Alzheimer's disease	Synapse	No relationship between PMI and synaptic volume.		No
Scheff 1993	https://pubmed.n	Human	Electron microscopy	Staining with osmium tetroxide, uranyl acetate, and lead citrate	3 to 13 hours	13 hours	20	Temporal lobe	Alzheimer's disease	Synapse	No relationship between PMI and the morphological features of synapses analyzed.		No
Schwab 1994	https://pubmed.n	Human	Light microscopy	Immunostaining for MAP1B, and MAP2, and tau	Comparison of surgical biopsy cases (n = 2) to autopsy cases (n = 5) with a PMI of 16 to 24 hours	24 hours	7	Temporal lobe	Biopsies from people with temporal lobe epilepsy	General neuronal immunostaining	In autopsy cases compared to biopsy cases, there was extensive perikaryal redistribution of MAP1B, and MAP2, and tau. However, this appeared to occur more slowly in human cases than rats.	"The distribution of tau-I, MAP2, and MAPIB in the surgical specimens was similar to that observed in the immersion-fixed rat brain, with tau-I being predominant in axons, MAP2 in dendrites, and MAP1B immunoreactivity present in both axons and dendrite. A slight perikaryal accu- mulation of MAP2 and MAPIB (Fig. Io), but not of tau-1, was evident. In the hippocampus obtained at autopsy, altera- tions in MAPIB and MAP2 were similar to those observed in the postmortem rat brain. Extensive perikaryal immuno- staining in the dentate gyrus, hilus, CA3, subiculum and occa- sional neuron in CA1 was observed in all autopsy tissues. Somatic tau-I immunoreactivity was observed in all of the autopsy cases, but in three ofthe cases (AI-A3) it was evident only in CA3c, adjacent to CA2 (Fig. 11). In the remaining cases, perikaryal tau accumulation was also evident through- out CAI, as well as in the dentate gyrus, hilus, CA3, and subiculum. This was particularly prominent in case A5, which had the longest postmortem interval." ... "Comparison of results in the rat with those in the human hippocampus obtained at surgery and autopsy demonstrated that postmortem alterations in tau, MAP2, and MAPIB in the human brain were similar to those observed in the rat, al- though they appeared to occur more slowly in the human"	Yes
Sele 2019	https://pubmed.n	Human	Light microscopy, electron microscopy	Morphological stain, including high-pressure freezing step	16 to 24 hours	24 hours	4	Frontal lobe	No known neurodegenerative disorder	Myelin and general cell morphology	On electron microscopy, the percentage of well-preserved myelin sheaths decreases with increasing PMI. On light microscopy, vacuoles increase with the PMI and there are decreasing structural details in the surrounding matrix with increasing PMI.	"Figure 3 shows that the PMI appeared to have an influence on the preservation of the myelin sheaths: the shorter the PMI, the higher the percentage of well-preserved parts of the myelin sheaths" ... "Light microscopic signs of autolysis were dilations around the vessels and the glia cells. These increased dramatically in size with increasing PMI (Fig. 5). The samples taken from case 1 (PMI 16 h, frontal grey matter) showed a dense organization of the neuronal tissue with a high number of finer structures. In the samples taken from frontal white matter (Fig. 5c-e), with increasing PMI, the organization of the tissue loosened more and more, and vacuole-like structures (putative degraded cellular compartments that could not be recognized any more) dominated instead (Fig. 5 and Fig. 6). Samples with a long PMI (case 4) showed an increased number of vacuole-like structures and hardly any structural details in the surrounding matrix"	Yes
Seppänen 2007	https://pubmed.n	Human	Light microscopy	H&E, collagen XVII immunostaining	48 to 120 hours	120 hours	11	Multiple regions	"[N]eurologically unimpaired subjects who had died from cardiovascular causes"	General neuronal immunostaining	No correlation between collagen XVII immunostaining and PMI.	"The expression of coll XVII was similar in the youngest and the oldest subject and no gender influence was seen. Furthermore, no difference in coll XVII-IR was seen in relation to post-mortem delay, which ranged from 48 to 124 h, or fixation time, ranging from 23 to 65 days. In line with this, statistical analysis did not reveal any correlation between coll XVII and the studied variables, i.e. age at death, gender, post-mortem delay and fixation time. However, an almost total lack of coll XVII-IR was noted in the one case that suffered pre-mortem cerebral hypoxia and that displayed numerous ischemic neurons in the brain samples (case no 11, rapidity of death Aiii)."	No
Sheedy 2012	https://pubmed.n	Human	Light microscopy	Morphological staining with H&E	3 to 72.5 hours	72.5 hours	105	Cerebellum	Not recorded	Cellular morphology	No correlation between histologic autolytic changes in the cerebellar granule cell layer and PMI.	Table 2	No
Sheng 1998	https://pubmed.n	Human	Light microscopy	IL-1α immunostaining	Average of 16 ± 2 h in those < 60 years old and 11 ± 2 h in those > 60 years old	Not recorded	22	Tissue blocks of hippocampus and adjacent mesial temporal structures	Neurologically normal individuals	Microglial morphology	No correlation between PMI and microglia morphology.	"These age-associated changes in microglial number and morphology were independent of postmortem interval"	No
Smith 1993	https://pubmed.n	Human	Light microscopy	Immunohistochemistry, "Synaptophysin-, serotonin-, cholecystokinin-, substance P-, and somatostatin-like staining"	5 to 21 hours, compared to surgically removed tissue	21 hours	9	Olfactory bulb	Multiple conditions, biopsies from people with epilepsy	General immunostaining	Compared to surgically removed samples, samples with longer PMI had deterioration, including diminished immunostaining of processes and the appearance of vacuoles. However, the immunostaining patterns were qualitatively similar.	"It is important to note that the general pattern and qualitative aspects of immunoreactivities were equivalent for the operating room and autopsy tissues. As expected, the longer postmortem intervals did result in progressive deterioration of the tissue. This included the appearance of vacuoles and a general decrease in the intensity and number of immunoreactive processes. Analyses of human tissue are limited due to the interval between cessation of respiration and fixation. This is especially problematic for studies using antibodies to antigenic sites that degrade very quickly prior to fixation. In these cases it is crucial to recognize that the absence of immunoreactivity does not necessarily imply the absence of the antigenic site. Consequently, it is appropriate to exercise caution in making comparisons with results obtained from other species in which perfusions were used to prepare the tissue. Nevertheless, the analyses and results described in the present report appear to be qualitatively consistent with prior studies."	Yes
Steiner 2006	https://pubmed.n	Human	Light microscopy	HLA-DR immunohistochemistry	8 to 72 hours	72 hours	32	Multiple regions	Schizophrenia	Microglial morphology	PMI correlated with ramified microglia cell morphology density in ACC/DLPFC and ameboid cell morphology density in hippocampus. No apparent adjustment for multiple comparisons. No correlation of microglial density with PMI.	"Postmortem interval showed a positive correlation with ramified cell density in ACC (left: r =0.47, P=0.01; right: r=0.48, P=0.01) and DLPFC (left: r=0.37, P=0.04; right: r=0.49, P=0.01) and ameboid cell density in left hippocampus (left: r=0.40, P=0.03)."	Yes
Stockmeier 2004	https://pubmed.n	Human	Light microscopy	Nissl staining	4 to 29 hours	29 hours	40	Hippocampus	Depression	General cell morphology	No significant correlation between PMI and neuron or glia cell size.	"When using Bonferroni corrections for multiple comparisons, there were no significant correlations between age, postmortem interval, tissue pH or brain weight, and the neuronal and glial density and size measures"	No
Sweet 2004	https://pubmed.n	Human	Light microscopy	Stained for Nissl substance with thionin	3.7 to 28.1 hours	28.1 hours	32	Primary auditory cortex	Schizophrenia	Neuronal morphology	No significant correlation between PMI and pyramidal soma volume.	"In the secondary model, the effect of diagnosis on ln(somal volume) was similar [...], without a significant effect due to subject age [...], gender [...], or PMI [...]"	No
Szocsics 2021	https://pubmed.n	Human	Light microscopy	Immunostaining for NeuN and parvalbumin and fluorescence microscopy	2 h 55 min to 5 h 5 min	5.083 hours	7	Primary motor cortex	"Control"	General neuronal morphology	No correlation between PMI and the soma size or density of Betz cells.	"Statistical analyses of soma size and density of Betz cells. Multiple regression analyses did not show significant changes by post-mortem interval (PMI)."	No
Tang 2001	https://pubmed.n	Human	Electron microscopy	Ethanolic phosphotungstic acid staining technique, modified for synapses in human autopsy brains.	Less then 60 hours	60 hours	5	Neocortex	No known neurological or psychiatric disorder	Synapse	When compared to expected electron microscopy findings, postmortem tissue up to 2.5 days is reported to have poor preservation, with debris, membrane fragments, and other problems, although they report it is still possible to count the number of synapses.	"The sIR was preserved for more than 96 hours postmortem, even in severely autolytic brains." ... "In cases of 24 to 48 hours of postmortem autolysis before fixation of the brain, the gray matter tissue showed only a mild diffuse loss of sIR, but the sharp definition in axons and around neurons was lost. The sIR remained reliable for routine autopsy examinations up to 72 hours between death and fixation of tissue, but sIR was lost in highly macerated brains with several days of autolysis." Also Figure 6.	Yes
Todtenkopf 1998	https://pubmed.n	Human	Light microscopy	Nissl staining, immunohistochemistry	6.5 to 42.5 hours	42.5 hours	25	Hippocampus	Schizophrenia	General neuronal immunostaining	No significant correlation in the density of GAD65-IR puncta on pyramidal neurons or non-pyramidal neurons with the PMI.	"Visual inspection of Nissl-stained sections indicated that there was good preservation of cellular and cytoarchitectonic detail following immunocytochemical processing" ... "As with age, there were no significant correlations of PMI with the density of GAD65-IR puncta on either PNs (r = 0.349, r = 0.138, r = 0.004, and r = 0.224, respectively) or NPs (r = 0.247, r = 0.145, r = 0.034, and r = 0.194, respectively). Similarly, when the regression analysis was performed separately for the two groups, no significant correlations were found". Figure 1 for representative images.	No
Tóth 2007	https://pubmed.n	Human	Light microscopy	Substance P receptor immunostaining	2 to >6 hours	6 hours	12	Hippocampus	Epilepsy	General neuronal immunostaining	No correlation between PMI and cell morphology, but decline in substance P receptor immunostaining after 6 hours of PMI, with fewer immunoreactive cells.	"In a preliminary experiment we examined 12 control brains with different post mortem delays and age in both genders. Although the general distribution and morphology of the cells was similar in each case, the long post mortem delay influenced the quality and quantity of immunostaining. The smallest number of cells was found in immersion fixed subjects with post mortem delays longer than 6 hours, therefore these control subjects were excluded from the present study"	Yes
Tóth 2010	https://pubmed.n	Human	Light microscopy	Immunostaining for calretinin	2 to 10 hours	10 hours	6	Hippocampus	"Control samples"	General neuronal immunostaining	Lower density of calretinin-immunoreactive cells in samples with long postmortem delay. Also shorter, varicose, and degenerating dendrites of calretinin-immunoreactive cells in samples with long postmortem delay.	"Significantly fewer calretinin-positive cells are present in every subregion in control samples with long post-mortem delay compared to controls with short post-mortem delay" ... "Our results suggest that these inhibitory cells in humans are also sensitive to ischaemic conditions and long post-mortem delay before fixation"	Yes

Study	Link	Species	General microscopy method	Visualization method specifics	PMI range	Longest PMI reported	Sample size	Brain region	Disease studied, if any recorded	Structural Feature	Outcome	Relevant text	Any significant/substantial correlation of a feature with PMI reported?
Uranova 2011	https://pubmed.n	Human	Electron microscopy	Osmium and uranyl acetate staining	Average of 5.9 ± 1.2 hours in control samples and 6.3 ± 1.8 hours in samples from people with schizophrenia	Not recorded	80	Prefrontal cortex	Schizophrenia	Myelin	No correlation between PMI and myelin morphometry.	"We did not find the effects of postmortem delay and neuroleptic exposure on the parameters of myelinated fibers." ... "We did not find the effects of postmortem delay and neuroleptic exposure on the frequency of myelinated fibers."	No
Vikhreva 2016	https://pubmed.n	Human	Electron microscopy	Staining with osmium tetroxide, uranyl acetate, and lead citrate	4.5 to 13 hours	13 hours	41	Prefrontal white matter	Schizophrenia	Oligodendrocyte size	No correlation of PMI with oligodendrocyte size.	"Correlation analysis showed no effects of age, postmortem interval, neuroleptic treatment and duration of disease on the oligodendrocyte parameters measured (all p > 0.2)."	No
Wegner 2006	https://pubmed.n	Human	Light microscopy	Nissl staining	16 to 96 hours	96 hours	18	Cortex	Multiple sclerosis	Neuronal morphology	No correlation between PMI and neuronal density, neuronal morphology, or glial density.	"Postmortem interval, formalin time, and age had no apparent effects on neuronal density, size, shape, or glial density."	No
Whitney 2008	https://pubmed.n	Human	Light microscopy	Morphological staining with Nissl and immunohistochemical staining for calbindin-D28k	3 to 48 hours (3 unknown)	48 hours	10	Cerebellum	Autism	Purkinje cell counting	No significant difference between PMI of cases with good and poor Nissl staining quality of Purkinje cells. Good reported calbindin staining of Purkinje cells in all cases despite a varying PMI.		No
Williams 1978	https://pubmed.n	Human	Light microscopy	Morphological stain, Golgi rapid preparation	30 minutes to 36 hours, and 2 biopsy specimens	36 hours	33	Cerebral cortex	"Normative human specimens"	General neuronal morphology	Increase in autolytic changes during the PMI, with lower quality rapid Golgi preparations after more than 6 hours postmortem, and worse quality of autopsy specimens compared to biopsy specimens.	"Golgi preparations from the 2 biopsy specimens of human neocortex were judged to be of excellent quality by all criteria. All the principal neuronal subclasses present normally in the various laminae of the cortex were well represented. A few scattered varicosities were present on the terminal portions of dendrites of some pyramidal cells. Autolytic changes observed in Golgi-rapid impregnations of the 29 autopsy cases after delays in fixation of 30 minutes to 36 hours after death are identical qualitatively to those observed in the cortex of mice after delayed fixation. These include variable completeness and selectivity of cellular impregnation, centripetally progressive dendritic deformities, changes in the hue of the background tissues from amber to yellow, and the appearance of granular precipitate in unimpregnated neurons."	Yes
Wu 2021	https://pubmed.n	Human	Light microscopy	Immunostaining for cocaine and amphetamine- regulated transcript (CART) and calbindin	14.1 ± 9.1 hours (for the control group, which is the only one with an observation reported)	Not recorded	15	Cerebellum	"Control"	General neuronal morphology	No correlation between the number of Purkinje cell somas or dendrites that a climbing fiber crosses with the PMI.	"In control cases, the number of PC somas or PC dendrites that a CF crossed did not correlate with PMI (P = 0.149 and 0.504, respectively), indicating that PMI is not a significant confounding factor."	No
Yoshida 2011	https://pubmed.n	Human	Light microscopy	Chromoganin A (CgA) immunostaining	0 to 3 days	72 hours	298	Hypothalamus	Multiple	General neuronal immunostaining	No correlation between PMI and immunostaining distribution for Chromoganin A.	"CgA was clearly detected in specific cell components: neurons in the hypothalamus (Fig. 3a), endocrine cells in the adenohypophysis (Fig. 3b), and chromaffin cells in the adrenal medulla (Fig. 3c). Cellular CgA immunopositivity in each tissue varied extensively among cases for each cause of death, without any relationship to the postmortem period, or age or gender of subjects." - Figure 3a as an example.	No